TRANSACTIONS

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OTOLARYNGOLOGY

JANUARY - FEBRUARY
1955

Volume 59 Number 1

ANNUAL MEETING, OCTOBER 9-14, 1955, CHICAGO

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*Menger, H. C.: New York State J. Med., in press.

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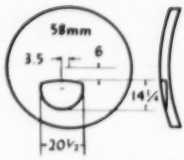

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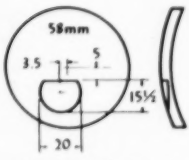

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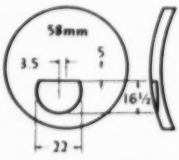

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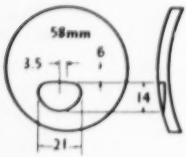

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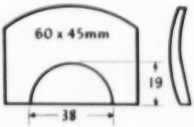

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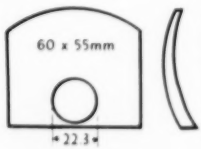

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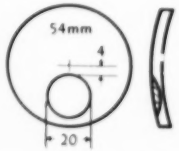

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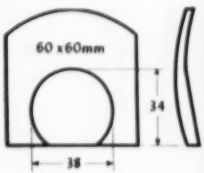

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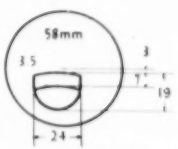

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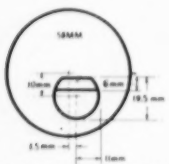
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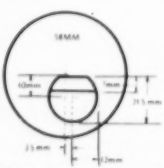
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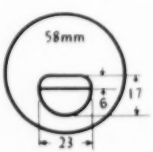

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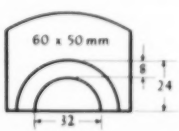

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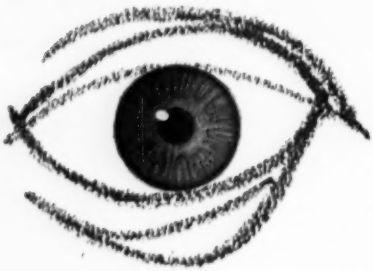

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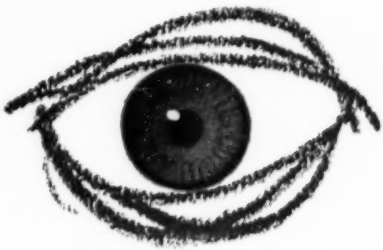

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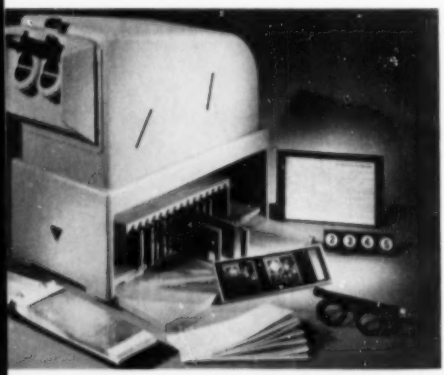

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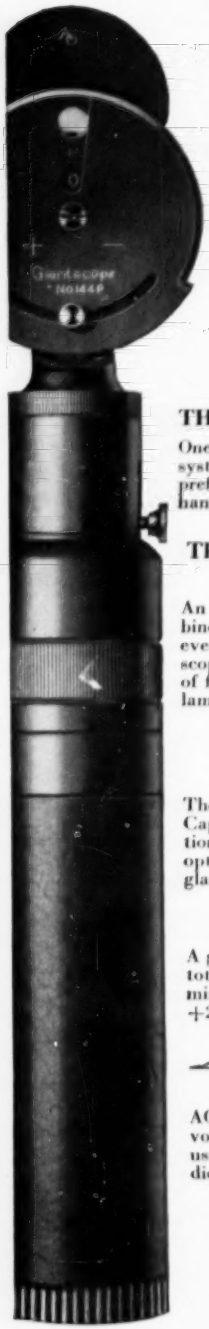

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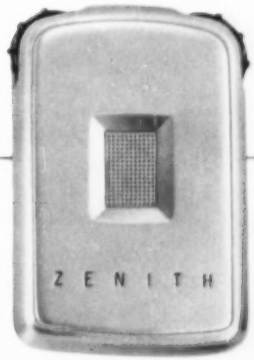

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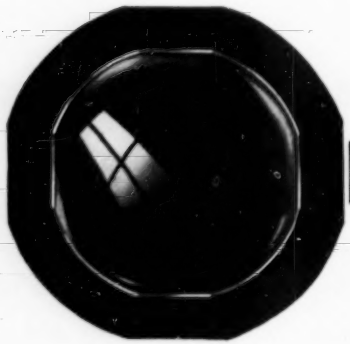

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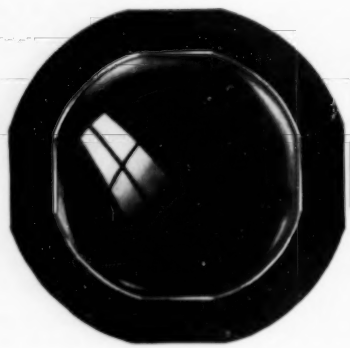

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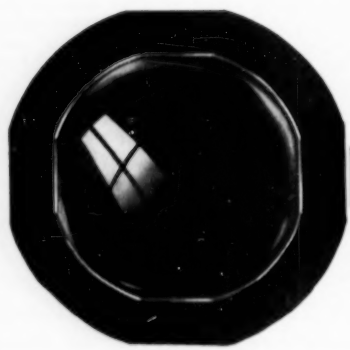

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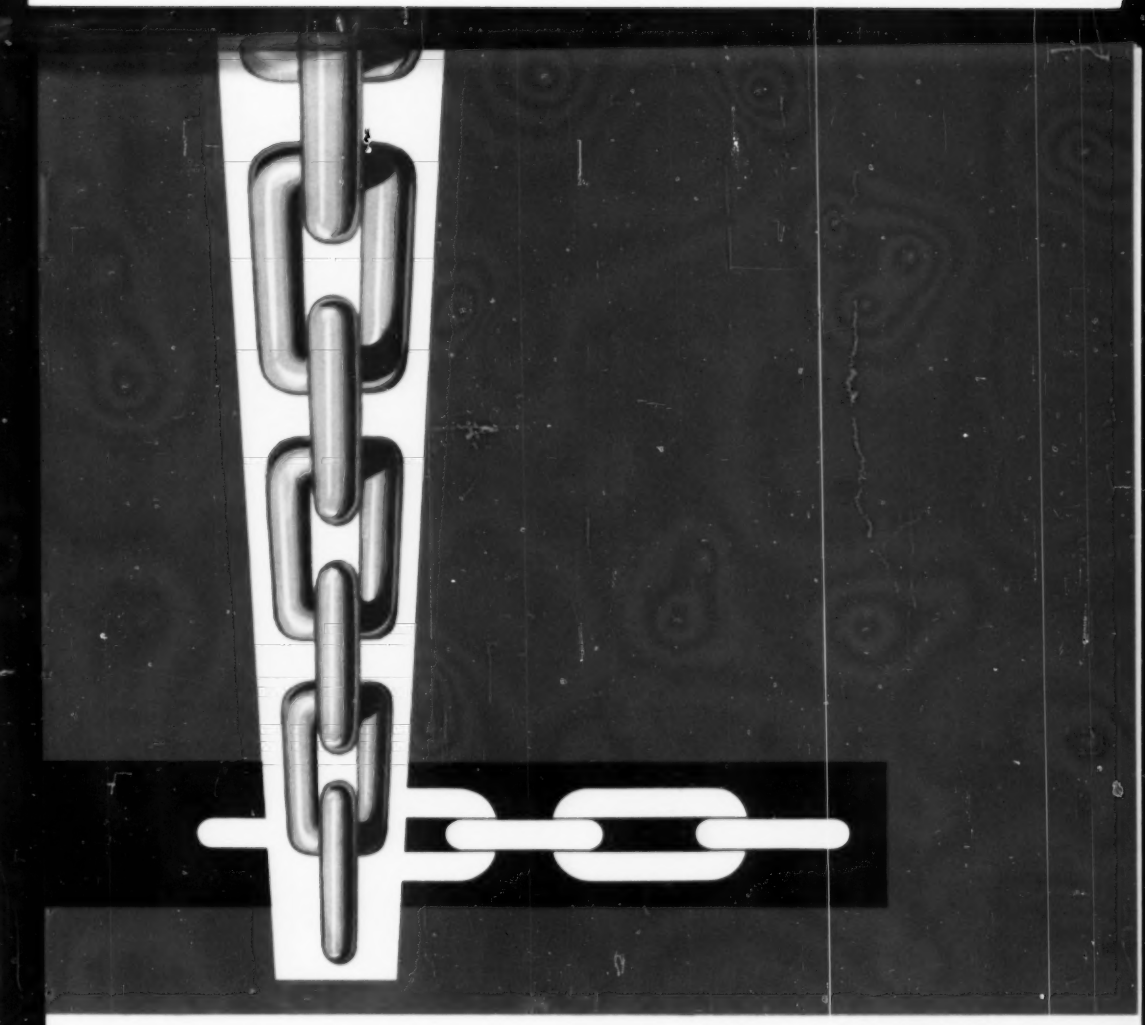

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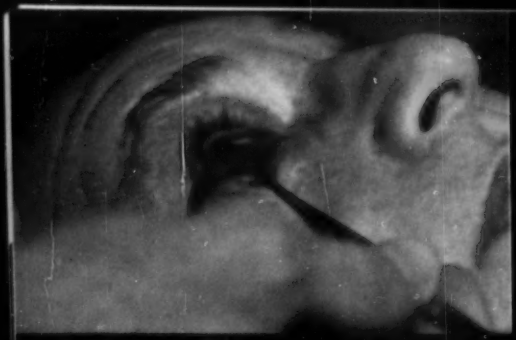
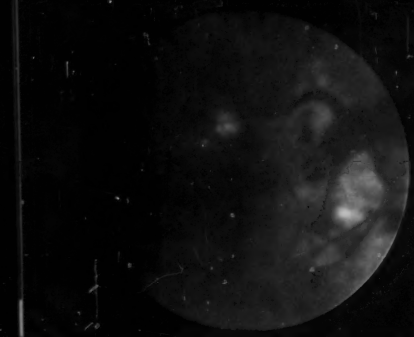
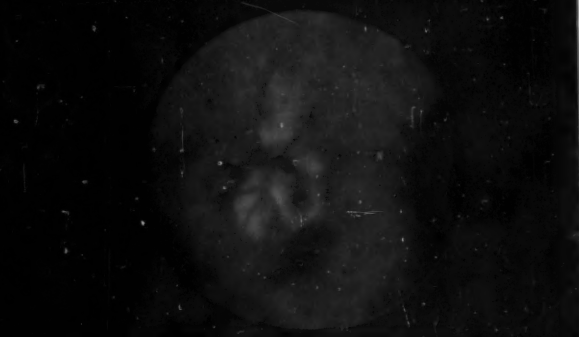


Welling up of blood upon incision of lid and conjunctiva, common in ophthalmic procedures, hampers surgery.

Minimal bleeding after Adrenosem therapy facilitates surgery and promotes healing.

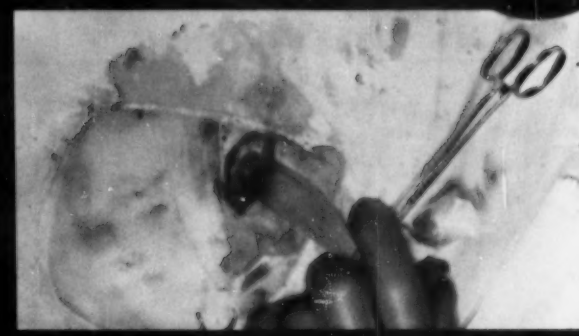


Anticipation of recurrent hemorrhage and visual defect is high in hypertensive patients.



After Adrenosem therapy. Note exudative areas. Maintenance therapy checks further hemorrhage.

Mastoidectomy with a bloody field obscures view, requires frequent suctioning.



After Adrenosem therapy. Note clearer operative field with minimized bleeding.

Multiple extraction without Adrenosem. Note seepage. Frequent suctioning is required.

After Adrenosem therapy. Note clearer field. Minimal bleeding facilitates surgery and promotes healing.

Adrenosem®

Salicylate *(brand of carbazochrome salicylate)*

Adrenosem is specific for conditions characterized by increased capillary permeability allowing oozing and seepage bleeding. To prevent seepage bleeding due to capillary permeability it is necessary to maintain tone of the capillary wall. This is supported by Pappenheimer's[1] work on capillaries. In cases of capillary permeability it is postulated that the connective material between the cells, which form the capillary wall, has lost its tone and allows the erythrocytes to pass through.

To confirm the action of Adrenosem in establishing capillary integrity the cheek pouch of a hamster was irritated with snake venom, which has the property of making the capillaries more permeable.[2] A great many petechiae appeared on the surface and a 500 X magnification showed the capillaries with extravasation of erythrocytes. Another animal was treated with Adrenosem, and 30 minutes later, the same amount of snake venom was used to irritate the cheek pouch. Microphotographs of the same 500 X magnification show the intact capillary walls and the actual blood cells coursing through the capillaries, without signs of escaping erythrocytes.

Fig. 1—*Trauma from snake venom produces hemoconcentration and stasis, indicative of altered vascular permeability.*

Fig. 2—*Trauma does not produce petechiae following Adrenosem injection.*

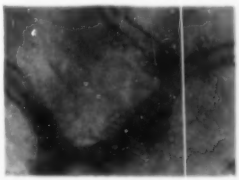


Fig. 1

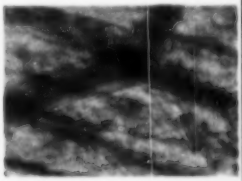


Fig. 2

Action

A systemic hemostat, Adrenosem restores capillary integrity by maintaining tone of intercellular tissue in the capillary wall. Adrenosem is unlike any hemostat available to medicine.[3,4] It does not interfere in any way with the blood clotting mechanism or its component parts. There is no sympathomimetic action and the blood pressure, cardiac rate and volume are not affected. Adrenosem may be administered simultaneously with any type of anesthetic, anticoagulant or vitamin K and heparin. Adrenosem will not interfere with the action of these agents.

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While the hemostasic action of epinephrine and related compounds has been recognized for a long time, certain characteristics limited their usefulness. A product of the metabolism of epinephrine seemed to offer the most promise. Given the name, Adrenochrome, this compound was studied extensively in both European and American centers. The handicap to wide use of Adrenochrome was its instability, so efforts were directed to find a stable modification that would retain the physiological activity. The answer to this proved to be the monosemicarbazone of Adrenochrome. Further research demonstrated that this compound could be made soluble enough to be practicable as an injectable solution, by transforming it to a sodium salicylate complex. This process has made possible Adrenosem Salicylate, a potent therapeutic agent which fills an important need in medicine.

1 *Pappenheimer, John R.: Passage of molecules through capillary walls, Physiol. Rev. 33:387-423, 1953.*

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4 *Roskam, J.: Arrest of Bleeding, Chas. C. Thomas, Publisher, Springfield, Ill., 1954.*

5 *Peele, J. G.: Adrenosem in the control of hemorrhage from the nose and throat (in press).*

6 *Bourgoyne, J. R. and Riddle, A. C., School of Dentistry, University of Tennessee: Personal communication, 1954.*

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Ampuls, 5 mg., 1 cc.: packages of 5.
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DOSAGE

- preoperatively to control oozing during surgery and provide clearer operative field: one to two ampuls (5 to 10 mg.) every two hours prior to surgery for two doses.
- postoperatively to prevent hemorrhage and check oozing: one to two ampuls (5 to 10 mg.) every two hours until there is no indication of undue bleeding. To maintain control, one ampul may be administered every three hours or 1 to 5 mg. orally t.i.d.
- to control active bleeding: one ampul (5 mg.) every two hours until bleeding is controlled; frequency of dose may then be diminished.
- to control mild, low-grade bleeding: one ampul (5 mg.) every three or four hours until bleeding is controlled; then 1 to 5 mg. orally four to five times daily until bleeding ceases.
- maintenance dosage to prevent bleeding in conditions where small vessel integrity may be impaired: 1 to 5 mg. orally t.i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally every three or four hours; if bleeding persists, oral dosage should be supplemented with one ampul (5 mg.) daily.
- pediatric dosage: up to four years of age—1 mg. intramuscularly or orally with same frequency as for adults, until bleeding is controlled. From four to twelve years of age—one-half the adult dosage as indicated.

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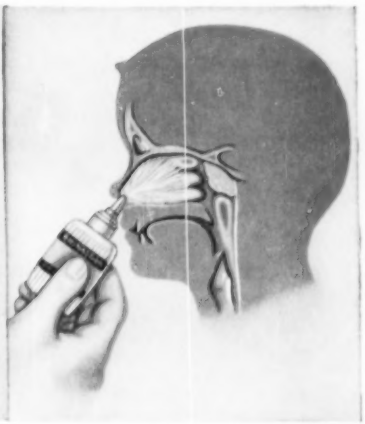

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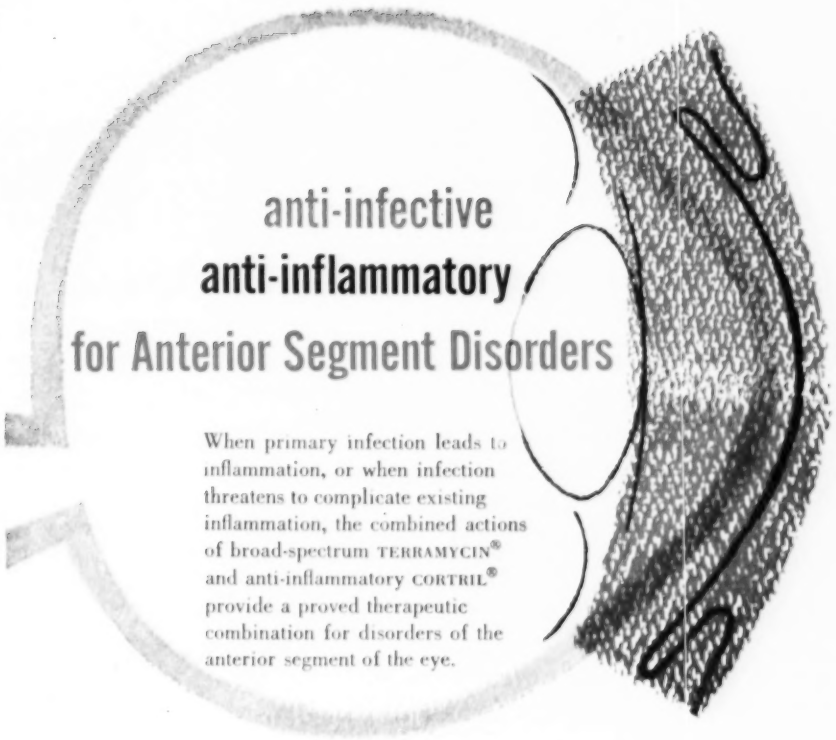

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Symposium: Retrolental Fibroplasia (Retinopathy of Prematurity)

The Academy's symposiums are not planned as a series of papers expressing individual views on a common subject. The object is rather to have a qualified team give their concerted appraisal of a given subject. The panel is supposed to consider and weigh the mass of material on the subject and then to present to you, or shall we say charge the jury with, what the members of the panel believe to be tenable.

The subject this year is retrolental fibroplasia. The men on this panel have had five sessions in which to discuss and to try to agree on controversial aspects. What will be presented here is not, therefore, the view of the individual participant but the consensus of the group. It is said that the pronoun "we" is properly used under three circumstances: (1) when royalty speaks, (2) when one is speaking in an editorial sense, and (3) when one harbors a tapeworm. I think we can add to this a fourth—when one speaks as a member of this panel.

We are using the term retrolental fibroplasia because it is so deeply entrenched in ophthalmological literature. We believe that the alternate term, retinopathy of prematurity, is a better one. Frequently the letters RLF will be used to refer to retrolental fibroplasia. Without further ado, therefore, we shall get on with the saga of retrolental fibroplasia.

ALGERNON B. REESE, M.D., *Moderator*
New York, New York

* * *

CLINICAL COURSE

WILLIAM COUNCILMAN OWENS, M.D.
WASHINGTON, D. C.

THE initial changes of retrolental fibroplasia are usually seen clinically during the first month after the birth of the premature infant. Only rarely does the onset of the disease appear as late as the tenth week of life. The early changes, however, may be overlooked unless repeated careful examinations of the fundus are made during the early postnatal period.

The active phase of the disease may be slow and smoldering or, in contrast, rapid and violent. It usually subsides by the time the infant is six months of age, when it is followed by the cicatricial phase. In the cicatricial phase, the eyes show residual manifestations of scarring, disorganization and destruction varying from clinically imperceptible changes to extensive atrophy. Thus, the clinical course of the disease may be divided into the *Active Phase*, the *Period of Regression* and the *Cicatricial Phase*.

THE ACTIVE PHASE

During the active phase, the disease can be divided into five stages.

Stage 1, The Vascular Stage

Usually the earliest change seen clinically is tortuosity and dilatation of the retinal vessels. In many cases, the veins become so large that they reach a size three or four times their normal diameter. The arteries may become so tortuous that they form sharp hairpin curves. In the periphery of the visible fundus, areas of neovascularization may be found. Usually the retinal vessels leading to such areas are somewhat larger than the dilated vessels seen in other areas of the retina. At the end of the vessels, fine, twig-like, delicate, newly

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formed vessels may be seen. Occasionally a transient narrowing precedes the dilatation of the retinal vessels.

Stage 2, Retinal Stage

As the disease progresses, the vitreous becomes hazy and the neovascularization becomes more profuse. Grey areas appear in the retina. These are usually located peripheral to the equator, but may be found in the posterior pole. In these grey areas, the retina appears elevated. Usually the vessels become obscure or, in some cases, even completely covered as they pass through the grey patches. Retinal hemorrhages may occur. They may be large but usually are less than one disc diameter in size. At this stage it is often difficult to see the details of the fundus because of the vitreous haze.

Stage 3, Stage of Early Proliferation

As the disease enters the third stage, fine strands of newly formed vessels with their supporting tissue can be seen extending like veils into the vitreous from localized areas of retinal elevation. Usually the elevated areas are found near the equator but occasionally may appear in the posterior fundus. In the milder cases only one area of proliferation may occur, but usually there are adjacent areas or perhaps several areas in different portions of the retina. Localized retinal detachment occurs in the periphery of the fundus.

Stage 4, Stage of Moderate Proliferation

With further progression more of the retina becomes involved. When the proliferation involves half the retina, the disease is said to have reached stage 4. The areas of activity may cover one side of the globe in a hemispheric fashion or, in some cases, the entire periphery may be involved, producing a detachment around the entire circumference of the retrolental space. During this stage the less severely involved portions of the retina may not be detached, although they may harbor areas of activity.

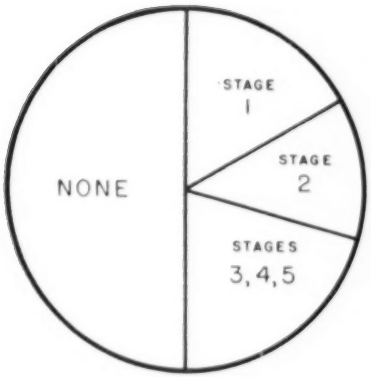
Stage 5, Stage of Advanced Proliferation

When the entire retina becomes detached, the disease has reached its most active stage. At this stage, occasionally a massive intraocular hemorrhage occurs, filling the entire vitreous.

PERIOD OF REGRESSION

When knowledge of the active phase of retrolental fibroplasia was new, the importance of spontaneous regression was not fully appreciated. Even in the early reports on the clinical course of the disease, occasional cases of spontaneous regression had been described. As more infants were studied during the early postnatal period, it became apparent that spontaneous regression was common. It is now known that the incidence of the active phase of the disease is much greater than had been expected from early reports which included only infants with severe residual damage.

Spontaneous regression from the active phase is characteristic of the disease. In the past acute retrolental fibroplasia occurred in about one half of all the premature infants in the lower weight groups. In the infants with active retrolental fibroplasia, spontaneous regression occurred at various stages. In about one third of them, the disease did not progress beyond stage 1. In another quarter of the infants with the active disease, regression occurred before the activity had proceeded past stage 2. In the remaining infants with active retrolental fibroplasia, stages 3, 4, and 5 were reached before regression occurred (fig. 1).



OCCURRENCE OF ACTIVE RETROLENTAL FIBROPLASIA

FIGURE 1.

In the cases with milder degrees of the active disease, recovery occurred with little serious destruction to the ocular tissues, while those cases with more intense activity usually developed extensive residua in the cicatricial phase of the disease. However, there is no certain way of determining during the active phase those cases in which regression will occur without serious destruction of ocular structures.

THE CICATRICIAL PHASE

The extent of residual destruction varies. In some cases, the eyes recover with no clinically visible changes. The others show an entire spectrum of residua extending from small retinal scars to the formation of a completely disorganized retrolental mass. The degrees of residual damage may be divided into five grades.

Grade I, Minor Changes

In the less severely affected eyes the fundus may be pale, or a slight decrease in the diameter of the vessels may be noted. Small areas of irregular retinal pigmentation occur, or occasionally a small mass of opaque tissue may be noted in the periphery of the fundus. These eyes are often myopic.

Grade II, Disc Distortion

In these cases the disc is pale and the vessels are pulled to one side. On the side opposite from the vessels, a pigment crescent is usually found around the margin of the disc. A small mass of opaque tissue remains in the periphery, toward which the vessels and disc are drawn.

Grade III, Retinal Fold

With more severe destruction, a retinal fold is found, extending to a mass of opaque tissue usually lying in the temporal periphery. The retinal vessels are incorporated in the folds. Usually there is only one fold, but occasionally in a single eye several folds are found, each extending to a peripheral mass of scar tissue.

Grade IV, Incomplete Retrolental Mass

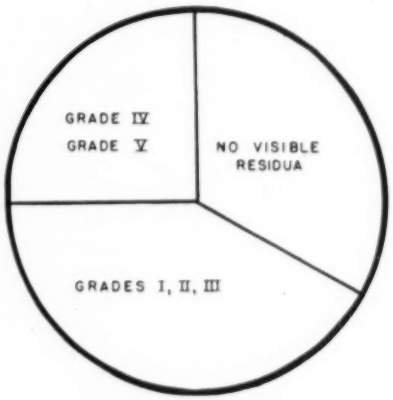
With more extensive destruction, a portion of the retina where proliferation has been marked becomes detached to form a retrolental mass. This may cover only part of the pupillary area. Through the remaining portion of the pupil, a red reflex may be obtained, and occasionally an attached portion of the retina may be seen.

Grade V, Complete Retrolental Mass

In the eyes that suffer the most severe damage, the entire retrolental space is filled with a mass of fibrous tissue containing the disorganized retina. In the periphery of the pupil elongated ciliary processes can be seen. The anterior chamber is characteristically shallow. Anterior and posterior synechias are usually present. The cornea may be-

come edematous if glaucoma occurs or if extensive anterior synechias form. When an eye is damaged to this degree, its normal growth is inhibited and it remains smaller than normal, giving the appearance of microphthalmos. The upper, and occasionally the lower, lid may be retracted at the palpebral folds. Enophthalmos may be present.

In the past only one quarter of the infants with active retrolental fibroplasia developed partial or complete retrolental membranes (grades IV and V). A third had minor retinal residua or small peripheral masses of tissue with retinal folds or distortions of the disc (grades I, II, III). The remaining infants recovered from the active phase of the disease with no visible residual changes (fig. 2).



RESIDUA FOLLOWING ACTIVE RETROLENTAL FIBROPLASIA

FIGURE 2.

COMMENT

Retrolental fibroplasia usually involves both eyes to the same degree. Occasionally the disease may be more advanced in one eye than the other, but it is rare to find cases with unilateral involvement. The continuous course of retrolental fibroplasia defies strict classification, for one stage blends into another and the clinical manifestations are almost legion. However, a classification of stages in the disease has been useful in spite of the continuous nature of the disease and its variety of clinical forms. The description of steps in the course of the disease aids in the comparison of data accumulated by different investigators in the attempt to determine the cause of retrolental fibroplasia.

Symposium: Retrolental Fibroplasia
(Retinopathy of Prematurity)

* * *

PATHOLOGY

JONAS S. FRIEDENWALD, M.D.
BALTIMORE, MARYLAND

THE histopathology of this disease can, I think, best be understood only in the light of our knowledge of the normal development of the retina. The retina is primarily neural tissue, and in the early embryological stages this developing organ is devoid of blood vessels. During this period the growing, differentiating structure derives its nourishment from the neighboring choroid and from the hyaloid vascular system of the vitreous.

At the 100 mm. stage capillary buds begin to sprout from the trunks of the hyaloid vessels as they pass through the optic disc. These sprouts invade the retina, spreading slowly peripheralward from the papilla. They are to be found at first mainly in the nerve fiber layer, and only in the later embryonal stages do vascular loops begin to penetrate the deeper retinal layers. These sprouting capillaries are accompanied by spindle-shaped cells which were formerly thought to be glial in origin but which Ashton has shown are mesenchymal. The vascular proliferation is normally most active in the sixth or seventh months, and it is babies who at premature birth still have their retinal vascularization to be completed that are susceptible to the disease. Evidence that incomplete vascularization of the retina in experimental animals is associated with susceptibility to the disease will be presented by Dr. Patz.

The first recognizable histologic lesion has to do with these sprouting capillaries. Instead of showing their normal growth extending smoothly throughout the retina, one finds in early stages of retrolental fibroplasia regions of avascularity associated with regions of patchy overgrowth of the retinal capillary endothelium. These patchy overgrowths produce localized whorls of varicose capillaries resembling the convoluted capillary tuft of a kidney glomerulus. Associated with these glomerular tufts there are patches of proliferation of the spindle-shaped mesenchymal cells formerly thought to be glial. Lesions of the type just described are to be seen as early as the second or third week of extra-uterine life and, in rare instances, even earlier. Morphologically similar lesions have been found in a few stillborn infants, but the relevance of these lesions to retrolental fibroplasia remains undetermined.

As the disease progresses, hemorrhages and transudations appear in relation to these abnormal vessels, and the vascular proliferation bursts out of the retina. Sheets of proliferating capillaries are often to be found on the vitreal surface of the retina and extending into the vitreous. Retinal detachment results in part from the transudative process, in part from the fibrosis of the vascular strands in the vitreous. The late cicatricial stages of the disease show little that is specifically characteristic, but the fibrotic remains of the so-called glomerular tufts are often still recognizable. These are of special interest to the pathologist because, when present, they may greatly assist in establishing the diagnosis.

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Symposium: Retrolental Fibroplasia
(Retinopathy of Prematurity)

PEDIATRIC CONSIDERATIONS

WILLIAM A. SILVERMAN, M.D.
NEW YORK, NEW YORK
BY INVITATION

COMPARISONS of premature infants, whether for the incidence of retrolental fibroplasia, the death rate, or any other purpose, are hampered by many difficulties. The most important difficulty is that of the range of diversity among these children. Although the accepted definition of a premature is "any newborn whose birth weight is less than 5.5 pounds," it is no exaggeration to say that from the standpoint of death risk a one-pound and a five-pound premature are as different from one another as a ten-year-old and an octogenarian.

The mortality at specific birth weights probably varies insignificantly from one part of the country to another. However, because of different admission policies the nurseries vary in composition, so that comparisons are as difficult to make as they would be between the death rate of a home for orphans and a home for the aged. The largest proportion of deaths that occur among premature infants occurs in the first hours and days following premature delivery, and the death rate quickly declines in the second and succeeding weeks.

The incidence of retrolental fibroplasia has been shown to be related to the degree of prematurity in a manner entirely similar to the death rate, so that geographical and temporal comparisons of the incidence of this disease have been hobbled by these same difficulties of dissimilarity from one nursery to the next.

During the past twenty years survival among premature infants has improved approximately 30 to 50 per cent. This fact is too insignificant to account for the increase in blindness among the survivors of premature delivery, which has been estimated to have increased by 500 to 1000 per cent.

It has been shown that the incidence of retrolental fibroplasia has varied from time to time in a manner which could not be explained on the basis of differences in the degree of prematurity of infants who are at risk.

From these considerations two basic recommendations can be made which should be followed by any clinical study of this disease. The first is that two or more groups should be compared only if the apportionment of the degree of prematurity is the same or after a suitable correction has been made; and the second is that two groups should be compared only in the same period of elapsed time. This last recommendation should be emphasized because of its particular significance.

If a theory concerning the cause of retrolental fibroplasia cannot be proven on laboratory animals, then the only alternative is a clinical trial. The hazards of a clinical trial are so well known and so especially applicable to this dis-

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ease that no re-emphasis should be necessary. If the new theory then is supported by clinical evidence which has been accumulated from the past or by comparison of one nursery or one period of time as opposed to another, then the best that may be said of this support is that it fails to disprove the theory. The argument that consistency of such post-hoc evidence constitutes proof is no more valid than the argument that consistent reports of flying saucers is proof that these objects are substantial.

This negative evidence should not be dismissed as wholly useless, for it is really important to emphasize that from this type of experience an excellent lead may be obtained, but it should not be mistaken as *proof* in the mathematical sense of the term.

Substantial positive proof, if it is to be dependent wholly upon the clinical trial, must be obtained by testing the theory, by a prospective study, using adequate controls, if it is to be deserving of consideration by exasperated students of this highly variable disease who have already heard too many cries of "wolf."

It is impossible to make any exact statement concerning the incidence of retrolental fibroplasia without the qualification of time and place. It is widely agreed, however, that the disease has increased radically in the past decade. Of prospective studies of incidence published to date, the majority agree that the incidence is related to the degree of prematurity. Both members of a fraternal twinship, both members of presumably identical twins have been involved. Siblings have been observed with this condition, and the rarity of this event would seem to reflect only the relatively rare occurrence of survival of two or more extremely premature infants born to the same mother.

Examples of retrolental fibroplasia have been seen in individuals of most of the major racial groups, and there have been no consistent sex differences reported.

I should like to discuss briefly some of the physiological considerations that distinguish prematurely born infants. I must first emphasize, however, that the most severe handicaps of prematurity are life-limiting, and among the survivors there is progressive improvement, so that the gap between the premature and the full-term infant is rather quickly closed.

First consider the blood. Among other chemical distinctions, fetal hemoglobin differs qualitatively from adult hemoglobin in its unusual ability to take up oxygen at low oxygen pressures. Although the blood of the premature contains relatively more fetal hemoglobin than that of the full-term infant, the order of magnitude is essentially the same—upwards of 80 per cent. The more striking difference is the slower rate of disappearance in the premature. Fetal hemoglobin is replaced in sixteen to twenty weeks in the full-term infant and a longer time is required for conversion in the premature.

The relationship of this fetal hemoglobin to the anemia of prematurity is not clearly defined. There is considerable evidence, however, that normal erythropoiesis is retarded, and this is supposedly the cause of extreme anemia of prematurity. The usual onset times of retrolental fibroplasia are earlier than the times of lowest blood values which are seen among these infants.

Although only widely discrepant figures are available, it is generally accepted that umbilical vein blood on its way to the fetus is approximately 50 per cent saturated with oxygen, and blood leaving the fetus on its way back

to the placenta contains quite low concentrations. This suggests good utilization of available oxygen.

After initial stabilization following premature delivery, only minimal reduction to approximately 90 per cent arterial oxygen saturation is seen when these infants are in atmospheric air. Despite this minimal reduction and with no evidence of cyanosis or distress, a high proportion of these infants exhibit irregular or periodic breathing. It was shown about ten years ago that with increasing oxygen enrichment to 25 per cent, and up to 85 per cent, concentration, increasing numbers of these infants could be made to breathe more regularly, with an increase in the minute volume, and to achieve 100 per cent saturation of arterial blood. It was generally accepted that despite minimal clinical signs, marginal anoxia existed, and this was the basis for the pediatric advice in the recent past that prematures remain in oxygen-enriched atmospheres for several weeks after birth.

The body temperature of the premature child is less stable under sudden environmental changes than is that of the child at full-term, and the premature remains poikilothermic for a longer period postnatally than the full term. Fat absorption is impaired, and for this reason low fat formulas have been generally, but not exclusively, used during the past decade. High doses, approximately twofold, of vitamin D reflected concern about the proneness of these infants to develop rickets. Vitamin C supplements are emphasized because of evidence that vitamin C permits the more complete metabolism of certain aromatic amino acids as well as for its antiscorbutic use.

Renal function is impaired because of the morphological immaturity of the glomeruli, which constitutes one of the major handicaps of this group. These infants excrete hypotonic urine and they have difficulty removing electrolytes from the plasma. However, after the early postnatal days prematures do not frequently exhibit any marked degree of edema while receiving the amount of electrolytes in cow's milk formulas although these formulas contain considerably more electrolytes than does breast milk.

Systemic manifestations of retrolental fibroplasia have not been uniformly reported and associated changes in other organs are denied by most students of this disease.

Studies available on mental measurements, and the largest published series is that of Dr. Samuel P. Hayes of Perkins Institution in Massachusetts, indicate that there is no difference between the retrolental fibroplasia blind and children blind from other causes. Moreover there is no evidence that the incidence of retardation is greater among children with retrolental fibroplasia than among prematures as a whole.

Symposium: Retrolental Fibroplasia
(Retinopathy of Prematurity)

ETIOLOGY OF RETROLENTAL FIBROPLASIA AND PRELIMINARY REPORT OF COOPERATIVE STUDY OF RETROLENTAL FIBROPLASIA

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The cause of retrolental fibroplasia has been the subject of many investigations since the disease was first described twelve years ago. Few congenital or environmental factors associated with either mother or infant have escaped consideration as possible etiologic agents.[17] Systematic examination of hospital records of mothers of premature infants has failed to bring to light any factors affecting the mother either before or during pregnancy, or at the time of delivery, which were consistently related to retrolental fibroplasia. Similar retrospective studies in relation to infants have shown a positive association between the incidence of retrolental fibroplasia and the administration of any of the following: water-miscible vitamins, iron, oxygen,[9] feeding of cow's milk,[6] added electrolytes,[7] and blood transfusions.[5] Experimental evidence has been reported indicating that the administration of alpha-tocopherol reduced the incidence of retrolental fibroplasia, suggesting that the cause of the disease might be a vitamin E deficiency in the infant[13]; other factors which at one time or another have been thought to be associated with retrolental fibroplasia include virus infection,[15] lack of hormones in the infant,[11] premature exposure of the infant's eyes to light,[8,12] and lack of vitamin A in the mother.[3,10] All of the factors mentioned above, except the administration of oxygen, have been largely eliminated as likely etiologic agents either through controlled clinical tests or further retrospective examination of records.

Confirmation of the observation that the incidence of retrolental fibroplasia increased with duration of exposure of

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infants to oxygen was reported from Australia and England by the fall of 1952.[2,4] The first controlled clinical test which subjected alternate infants to varying amounts of oxygen had just provided strongly suggestive indications that the search for a causative agent for retrolental fibroplasia might be at an end.[14] Early confirmation or denial of these data was urgently needed, especially in view of the bizarre fluctuations in incidence of retrolental fibroplasia noted previously with studies on other factors.[17] This variability of the incidence both temporally and geographically, indicated the need for skepticism in evaluation of most published investigations. One could anticipate as provocative a discussion from the statement that oxygen was probably a factor in production of retrolental fibroplasia as from one suggesting that it was unrelated to the disease. Moreover, many investigators in the field could recall that the results of administering alpha-tocopherol or cortisone[16] were as striking as those obtained by lessening the exposure to oxygen, but that subsequent work failed to confirm the initial observations. Reported animal experimentation with respect to oxygen in 1952 was as much concerned with the effects of hypoxia as hyperoxia, and the results obtained were either negative or appeared to relate so indirectly to clinical experience as to remain unconvincing.

In the midst of this furor, at the 1952 meeting of the American Academy of Ophthalmology and Otolaryngology the idea of organizing a National Cooperative Study on Retrolental Fibroplasia was conceived. Under the auspices of the National Institute of Neurological Diseases and Blindness of the U. S. Public Health Service a meeting of ophthalmologists and pediatricians interested in the problem was held in Bethesda, Maryland, to discuss further the advisability of setting up an organization to evaluate on a national scale any promising lead for the solution of the retrolental fibroplasia problem. As a consequence of this, and several other conferences, investigators from eighteen hospitals agreed to pool their nursery facilities and interests, and join in a cooperative study to determine the degree of association between the incidence of retrolental fibroplasia and the administration of oxygen. This investigation anticipated the opportunity of obtaining information of a general nature concerning the premature infant and his parents, which might be helpful not only in interpreting the results with respect to the use of oxygen but in formulating future investigations. Furthermore, this study would also make possible the determination of whether the survival of the premature infant was dependent on the amount of oxygen given him.

Because of the immediate practical importance of the results obtained to date, the steering committee of the Cooperative Study thought it expedient to present a preliminary report of the observations made for the first six months of the investigation, which began July, 1953.

Simply stated, the primary aim of the investigation was to compare the incidence of retrolental fibroplasia in two groups of premature infants, which were to receive oxygen for different periods of time but otherwise would be maintained under as nearly similar conditions as possible. The first group was to receive oxygen in concentrations of over 50 per cent for 28 days, a procedure which was then considered essentially routine in view of the degree of prematurity of the infants involved, and a second group which was to receive either no oxygen or limited amounts prescribed only on the basis of clinical urgency. These groups will be referred to as routine and curtailed, respectively. The study was to involve only those in-

fants who survived forty-eight hours. In order to concentrate efforts on infants where the incidence might be expected to be the highest, only those infants having birth weights of 1500 grams or less were admitted to the study. Infants within each individual hospital were to be assigned to the groups on a statistically random basis.

In planning this clinical trial, consideration was given to both the possibility that the administration to infants of substantially lesser amounts of oxygen than was customary might significantly increase mortality, and that the continued use of the customary amounts of oxygen might result in an unnecessarily high incidence of the disease.

This Scylla and Charybdis dilemma was resolved by a design in which but one twelfth of the total number of infants expected to be in the study was to be assigned to the routine oxygen group. In this way, should oxygen indeed prove to be positively associated with retrolental fibroplasia, the number of infants in the participating hospitals who would receive routine oxygen during the ensuing year would be substantially less than normal.

To obtain an answer to the questionable role of oxygen as soon as possible, whilst not materially sacrificing temporal controls, one infant was to be assigned to the routine oxygen group for every two babies assigned to the curtailed oxygen group during the first three months of the study. If at the end of the three-month period there was no difference in mortality, all babies were to be assigned to the curtailed group until such time as the incidence of retrolental fibroplasia in the two groups could be evaluated. This method of allocating infants provided an even greater safeguard with respect to mortality, since at the end of the first three months, a sufficient number of infants would already have been accumulated in each group to permit a decision as to whether the mortality rates were different.

Because the susceptibility of infants to retrolental fibroplasia is thought to vary inversely with their birth weight, assignment of infants to the routine or curtailed groups was made within three weight classifications (1000 gm. or less, 1001-1250 gm. and 1251-1500 gm.). This procedure increased the certainty that the distribution of susceptibility of the infants in the curtailed and routine oxygen groups would be essentially the same.

The concentration of oxygen was to be measured three times a day after the baby was admitted to the study. The infant receiving routine oxygen was to be returned to an air environment at or after twenty-eight days by reducing the flow rate of oxygen by one third over each of three successive days.

RESULTS

Of the 700 or more infants who were entered in the Cooperative Study during the course of the year, this report will refer only to those who were admitted to the study between July 1, 1953, and January 1, 1954. The data concerning mortality refer to all infants admitted to the study who survived 40 days. Those concerning ocular findings relate to infants whose eyes have been followed for two and a half months or longer, approximately 98 per cent of all the infants surviving to this age.

Since all infants assigned to the routine oxygen group were born during the first three months of the study, comparison of the incidence of retrolental fibroplasia in this group should be made only with those infants assigned to the curtailed oxygen group who were born during the same time period if strict temporal control is to be maintained. However, the incidence rates in the cur-

TABLE I

PERCENTAGE MORTALITY

	INFANTS			
	TOTAL	DIED	LIVED	PERCENTAGE MORTALITY
Routine O_2 group (3 mo.)	68	15	53	22.1
Curtailed O_2 group (1st 3 mo.)	144	36	108	25.0
Curtailed O_2 group (6 mo.)	323	65	258	20.1

Routine O_2 group vs. Curtailed O_2 group (first 3 months) $P = > 0.5$
Routine O_2 group vs. Curtailed O_2 group (6 months) $P = > 0.8$

tailed groups during the first three months and the second three months do not differ significantly, so that for the purposes of this preliminary report it seems appropriate to consider all of the infants in the curtailed group during the first six months together. Hence, the data in the tables will refer to infants during the whole six-month period, and presentation of data for each three-month period will be deferred until a complete report is written.

Results of ophthalmological examination were classified according to numerical evaluation of active and cicatricial grades of retrolental fibroplasia described by Drs. Reese, King and Owens.[1]

MORTALITY

Of the 68 babies assigned to the routine oxygen group (from age 48 hours), 15 succumbed prior to 40 days, giving rise to a mortality of 22 per cent; of the 144 babies in the curtailed oxygen group during the same time period (the first three months only), 36 succumbed, giving a mortality rate of 25 per cent (table I). This difference in mortality rate is not significant as it could be expected to occur by sampling variation. During the first six months 323 babies were assigned to the curtailed group, 65 of whom succumbed, representing an overall mortality of 20 per cent in this group. This rate is likewise not significantly different from that found in the routine oxygen group.

The difference in distribution of the infants with respect to the number of days in oxygen is apparent (fig. 1). The average stay in oxygen in the curtailed group was five days compared with an average stay of thirty days in the routine group.

The percentage of infants who developed active stages of retrolental fibroplasia, with the maximal stage developed in the poorer eye used as the basis for computation, is shown in Table II. Of the 245 infants in the curtailed oxygen group, 30 per cent showed

TABLE II

PERCENTAGE OF INFANTS WHO DEVELOPED ACTIVE STAGE RLF
(Maximal Stage Reached)

STAGE	ROUTINE O_2 (3 mo.) (53 Infants) PER CENT	CURTAILED O_2 (6 mo.) (245 Infants) PER CENT
Normal	**28**	**70**
1	25	15
2	23	8
3	13	5½
4	9	1
5	2	½
Total 1 - 5	**72**	**30**

$P = < 0.01$

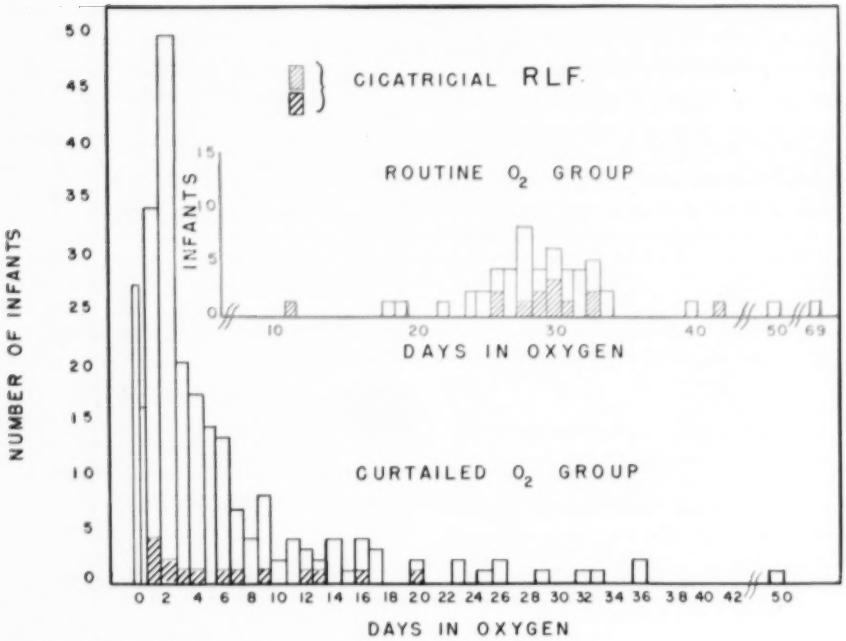

FIG. 1—Distribution of the infants according to the number of days in oxygen for the routine and curtailed oxygen groups. The number of infants is shown on the ordinate and the number of days in oxygen on the abscissa. The blocks refer to the infants in the curtailed oxygen group, and the inset upper blocks to those in the routine oxygen group. The crosshatched area represents the number of infants whose eyes developed some cicatricial grade of retrolental fibroplasia.

some active stage of retrolental fibroplasia compared with 72 per cent of the 53 infants in the routine oxygen group. This difference between the groups is significant at the 1 per cent level.

Of the 245 infants in the curtailed group 15, or 6 per cent, showed some cicatricial grade of the disease compared with 13 of the 53 infants, or 25 per cent, in the routine oxygen group (table III). Again the incidence of ocular change is significantly greater in the group which received the routine oxygen.

The manner in which the cicatricial cases of retrolental fibroplasia in both the routine and curtailed oxygen groups

TABLE III

PERCENTAGE OF INFANTS WHO DEVELOPED CICATRICIAL GRADE RLF

	ROUTINE O_2 (3 MO.) (53 Infants)	CURTAILED O_2 (6 MO.) (245 Infants)
GRADE	PER CENT	PER CENT
Normal	**75.6**	**93.8**
I	5.7	1.6
II	3.8	1.6
III	3.8	0.8
IV	3.8	1.6
V	7.5	0.4
Total I - V	**25.0**	**6.0**

$P = < 0.01$

TABLE IV

DISTRIBUTION OF CICATRICIAL GRADES RLF, AND AVERAGE NUMBER OF DAYS IN OXYGEN BY WEIGHT GROUP

WEIGHT GROUP GM.	AV. NO. DAYS IN OXYGEN	NORMAL	RLF	PER CENT WITH RLF
1000 or less	12	31	2	6
1001 - 1250	14	90	13	14
1251 - 1500	10	149	13	9

were distributed with respect to duration of oxygen is shown in figure 2. Despite the fact that incidence rates of the disease are significantly different between the routine and curtailed oxygen groups, some cases of retrolental fibroplasia occurred in infants throughout the whole range of stay in oxygen. The significance of this will be discussed subsequently.

Table IV shows the distribution of infants in each of three weight groups who developed cicatricial grades of retrolental fibroplasia where the poorer eye is again used as a basis of computation. The table also shows the average number of days in oxygen for each weight group. In this series of cases there was no significant difference in incidence between the three weight groups, or in the amount of oxygen they received. While the number of cases is insufficient to contest effectively the generally held belief that lighter infants are more susceptible to retrolental fibroplasia, they are included here because they provide comparison of the average length of stay in oxygen. Additional data from the Cooperative Study should help clarify this question of possible differences in susceptibility among premature infants *having birth weights of 1500 grams or less.*

The percentage of the infants who developed the active stages of the disease but whose eyes eventually showed no cicatricial retrolental fibroplasia is shown in table V. The percentage of regression clearly decreases with increasing severity of the active phase. The observation that the eyes of nine tenths of the infants reaching active stages 1 or 2, and approximately one half of those reaching stage 3 regressed to normal indicated the favorable prognosis during the early stages of retrolental fibroplasia.

The relation between the incidence of cicatricial retrolental fibroplasia and the number of days the infants were in oxygen after entry into the study (age 48 hours), was computed for all the infants, independent of the group to which the infants were assigned by protocol (fig. 2). The incidence of retrolental fibroplasia for the first week was computed on a daily basis (filled circles), but because of the lesser number of infants whose exposure exceeded one week, the incidence thereafter was

TABLE V

PERCENTAGE OF ACTIVE STAGES OF RLF WHICH REGRESSED TO NORMAL

MAXIMAL ACTIVE STAGE	REGRESSION TO NORMAL
1	90 (45/50)
2	87 (27/31)
3	45 (9/20)
4	25 (2/8)
5	0 (0/2)

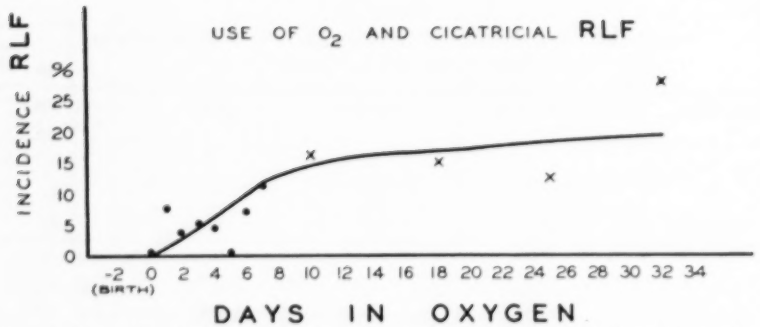

FIG. 2—The relation between the incidence of cicatricial retrolental fibroplasia and the number of days the infants were in oxygen after entry into the study. Results of analysis of the data using methods designed to evaluate the effect of cumulative experience are indicated by the curve.

calculated on a weekly basis (crosses). With few exceptions the exposure to oxygen occurred immediately upon entry into the study, and the incidence of retrolental fibroplasia was zero in those infants who received no oxygen after entry into the study. There were 43 infants in this group. Thereafter, the risk of developing retrolental fibroplasia appeared in this analysis to increase with exposure to oxygen during the first ten days of life. This preliminary observation is similar to results obtained in animal experimentation and may prove to be of considerable importance. It suggests first that the mechanism involved is of the type of an all or none insult to the eye which is associated with relatively short exposure to oxygen in the early days of life, and secondly, the necessity of restricting the use of oxygen even during the first week of life.

DISCUSSION

The positive association between the incidence of retrolental fibroplasia and administration of oxygen would not in itself indicate that oxygen is the cause of the disease, for it may be that oxygen is simply associated with the true cause. Previous investigators[18] have also been concerned with this problem and have pointed out that while oxygen therapy and retrolental fibroplasia are related to each other, they may both be dependent upon such common factors as illness or birth weight.

The random assignment of infants proportionately by birth weight to the two types of oxygen regimens in this study, and the subsequent finding of significant differences in incidence of retrolental fibroplasia, indicate that weight at birth did not seem to be a controlling influence in development of the disease, and similarly, illness is not likely to be the controlling influence because the number of babies who might be expected to become ill should be the same in both groups. However, it is possible that other unrevealed factors associated with oxygen therapy may, either alone or together with oxygen, produce the disease.

The study was designed primarily to evaluate the effect of duration of exposure of the infants to oxygen with regard to the incidence of retrolental fibroplasia. However, data collected from the study allow a certain amount of analysis of the effect of concentration. For example, the average concentration of oxygen to which the infants were exposed during the first nine days was 53 per cent in those cases developing either

active or cicatricial retrolental fibroplasia, whereas the average concentration for those infants whose eyes were normal was 47 per cent. This suggests that there may be a critical concentration beyond which the risk of retrolental fibroplasia rises rapidly, or conversely stated, the use of oxygen below certain minimal concentrations may involve less risk of retrolental fibroplasia. Further analysis of the data with regard to concentration is contemplated and will be reported later.

The observations reported in this study bear indirectly on the question of slow or rapid weaning of the infant from oxygen. Since the incidence of retrolental fibroplasia appears to increase with each additional day of exposure to oxygen during the first week or ten days of life, it follows that prolonging the stay of the infant in oxygen to permit slow weaning may increase the risk of developing the disease. Further analysis of the data when they become available may make it possible to evaluate the relative risks involved.

SUMMARY AND CONCLUSIONS

Hypotheses of the etiology of retrolental fibroplasia, other than those concerned with the use of oxygen, have failed to receive confirmation. Retrospective studies initially suggested that the incidence of the disease was correlated with exposure of the premature infant to oxygen. A study using alternate controls provided more convincing evidence of the relation between the use of oxygen and retrolental fibroplasia.

A national Cooperative Study involving 18 hospitals was designated to evaluate quickly in a controlled clinical trial not only the association between the use of oxygen and the incidence of the disease but the mortality risk involved in the use of drastically curtailed amounts of oxygen.

Premature infants who weighed three pounds and five ounces, or less, at birth and who survived 48 hours were randomly assigned to one of the following two regimens of oxygen administration:

1. A routine oxygen group which received approximately 28 days of oxygen at concentrations of at least 50 per cent
2. A curtailed oxygen group which received oxygen only on the basis of frank clinical need

All other conditions concerning the care of the infants were kept as constant as possible.

This report which, it must be emphasized, is only of a preliminary nature, shows that there was no significant difference in mortality of infants in the two groups. The incidence of both the active and cicatricial forms of retrolental fibroplasia, however, was significantly higher in the group which received routine oxygen.

The incidence of the cicatricial form of retrolental fibroplasia has thus far been found to increase with the duration of exposure of the infants to oxygen during the first ten days after entry in the study, but to increase little more with additional exposure. These observations suggest that some initial insult occurs to the eyes which is associated with relatively short exposures to oxygen.

The data indicate that (1) limiting the amount of oxygen used in the care of premature infants to that required for clinical emergency is without effect on the survival rate of the infants; (2) the incidence of retrolental fibroplasia is positively associated with the use of oxygen; and (3) much of the deleterious effect appears to be associated with exposure to oxygen within the first ten days of life.

Therefore, it is concluded that pediatricians should restrict the use of oxygen to those minimal amounts which on the basis of frank clinical need are required for the survival of the infant. The investigators responsible for this study hope that its continuation during the current year may establish relatively safe levels for such oxygen therapy.

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Symposium: Retrolental Fibroplasia (Retinopathy of Prematurity)

EXPERIMENTAL STUDIES

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THE first attempts to reproduce retrolental fibroplasia (RLF) in animals were made originally by Terry,[17] who conducted experiments on pouch opossums and newborn rats with measures calculated to cause precocious closure of the hyaloid artery. Dr. Terry did not elaborate on the techniques of his experiments; however, he concluded that the results were completely negative.

Hepner[10] reported the production of a condition similar to retrolental fibroplasia in kittens. Here newborn kittens were given enormous quantities of fluids with high electrolyte content and large blood transfusions until the animals were completely waterlogged with massive edema. Hepner's lesions, however, consisted only of dilatation of the retinal vessels with areas of retinal edema. It is apparent that the retina here simply participated in the generalized edema. The extra-retinal vascular proliferative changes of the disease were not observed. Hepner's experiments, however, raise the possibility that high electrolyte feedings in infants might increase the tendency for retinal edema in RLF.

Callison and Orent-Keiles[3] reported intraocular hemorrhages and disturbance of the vitreous, suggesting retrolental fibroplasia, in vitamin E deficient young rats. Working with Callison in her own laboratory and using the identical technique that she reported, we have been unable to reproduce the lesions that were originally described.[15]

Ingalls and co-workers[11] observed fetal anomalies in a very small percentage of mice exposed to an anoxic environment in utero. These changes consisted primarily of hyaloid system abnormalities and the experiments were conducted considerably before retinal vascularization had started. It is, therefore, doubtful that these lesions can be related to classic human RLF.

Campbell[4] using rats, Ashton[1] kittens, and our group[14] mice and rats, have observed that an anoxic environment causes a slightly abnormal pattern of retinal vascularization with a denser than normal capillary network and narrowing of the capillary-free zone about the arterioles. Typical vaso-proliferations of RLF erupting into the vitreous were not detected. These data combined with studies on high oxygen implicate a relative oxygen lack in the retina as an important stimulus to vascularization.

There are now available experimental studies on high oxygen administration from three independent laboratories. Gyllensten and Hellstrom[7] in Stockholm observed intermittent intraocular hem-

From the Departments of Ophthalmology and Pediatrics, District of Columbia General Hospital, and the Hoffberger Research Laboratories, Sinai Hospital, Baltimore, Maryland. These studies were aided by grants from the National Institute of Neurological Diseases and Blindness, Public Health Service, and the National Society for the Prevention of Blindness.

Presented at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

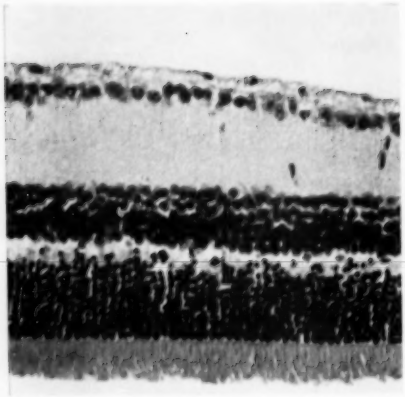


FIG. 1—Cross section of retina of normal 21-day-old mouse. Note relatively acellular character of the nerve fiber layer. (Hematoxylin-eosin. X400.)

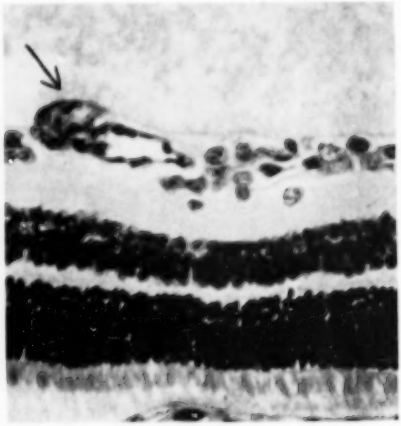


FIG. 2—Section of retina of 21-day-old mouse raised in 70 per cent oxygen since first week of life. Arrow points to abnormal proliferating endothelial nodule in the nerve fiber layer. (Hematoxylin-eosin. X400.)

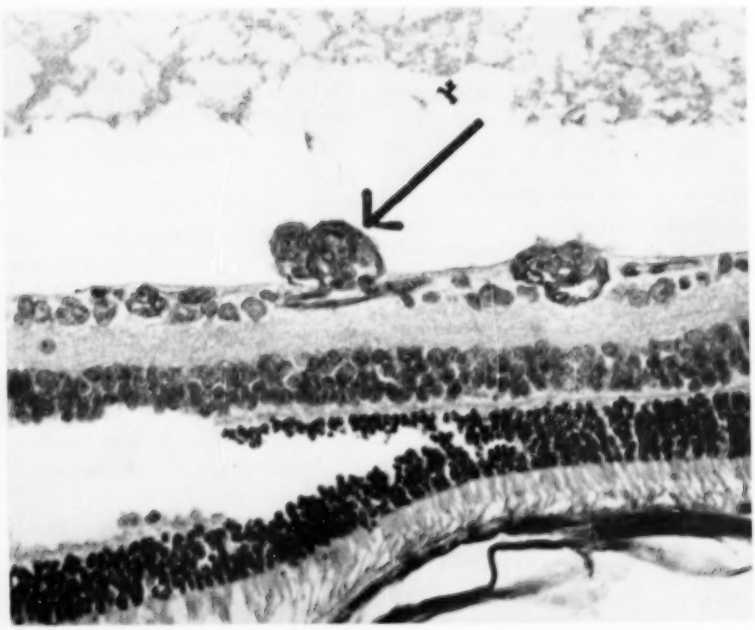


FIG. 3—Section of retina of 21-day-old rat raised in 70 per cent oxygen since first week of life. Arrow points to abnormal proliferating endothelial nodule in the nerve fiber layer. (Hematoxylin-eosin. X400.)

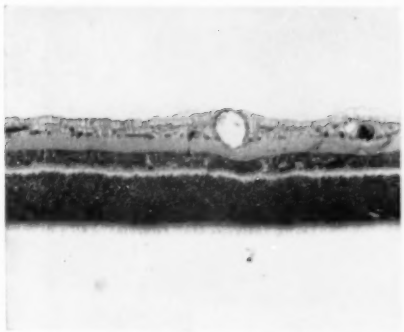


FIG. 4—Cross section of retina of normal 21-day-old kitten injected with India ink. Note that capillaries are confined to the retina. (Hematoxylin-eosin. X300.)

orrhages, vitreous degeneration, and abnormal proliferation of the hyaloid vessels in mice exposed to 100 per cent oxygen. In a later publication[8] they cite the typical vascular proliferative lesions of RLF. Ashton and co-workers,[1,2] in carefully conducted experiments on kittens, have observed the characteristic retinal vascular lesions described in human retrolental fibroplasia and have contributed greatly to our understanding of the mechanism of oxygen action in RLF. In our own laboratory, experiments on the ocular and systemic effect of high oxygen were instituted to parallel a controlled nursery study that was in progress in our hospital. Where newborn or young mice, rats, kittens and puppies were exposed to high oxygen, lesions typical of early human retrolental fibroplasia were observed (figs. 1-8).[6,9,16]

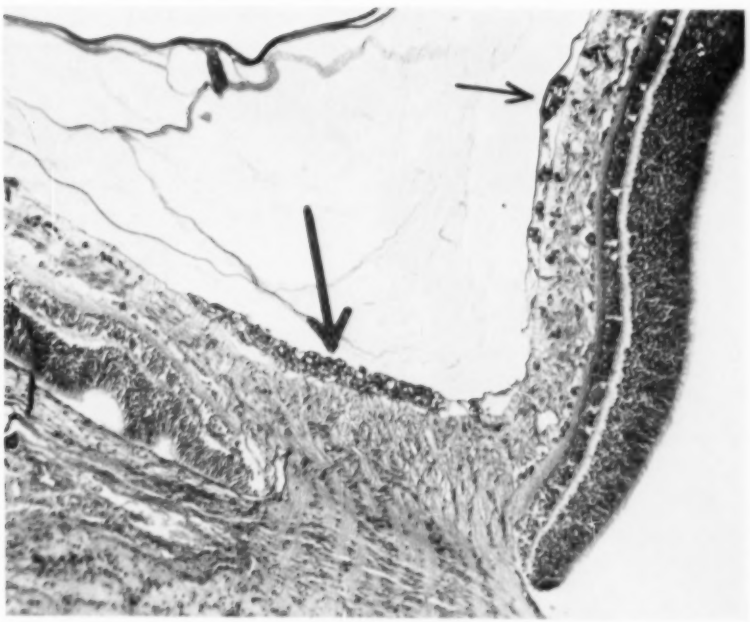


FIG. 5—Section of 28-day-old kitten retina. The animal was raised in 70 per cent continuous oxygen from the second day after birth. Large arrow points to abnormal proliferating capillary buds on surface of retina and small arrow indicates neovascular membrane over optic nerve. (Hematoxylin-eosin. X100.)

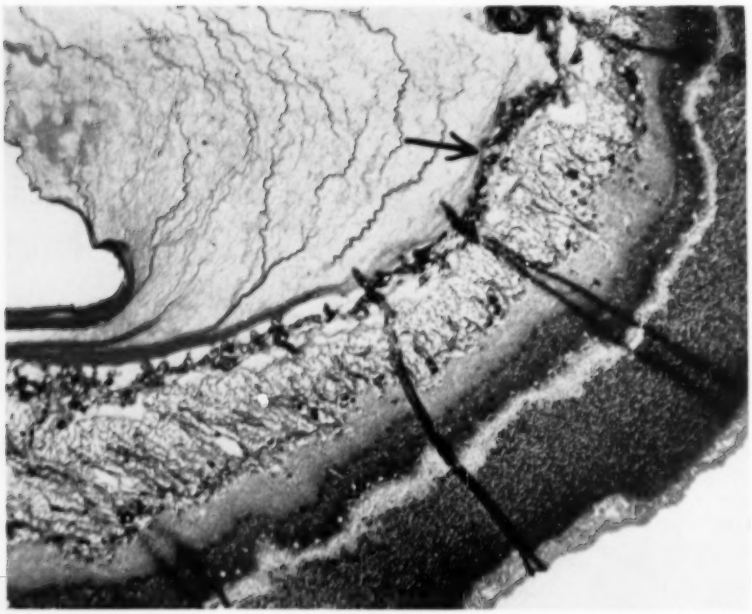


FIG. 6—Cross section of eye of 21-day-old kitten who received oxygen first week of life. Note capillary growth on vitreal surface of the retina. (Hematoxylin-eosin. X100.)

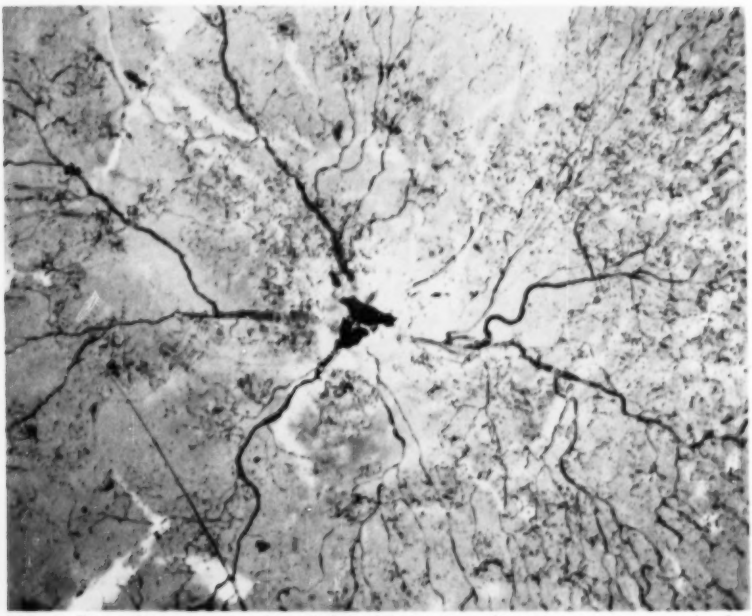


FIG. 7—Flat retina preparation from 33-day-old kitten exposed to 70 per cent oxygen for first week of life, then removed to room air. Note abnormal pattern of retinal capillaries with numerous tuft formations near disc. (Unstained preparation. X20.)

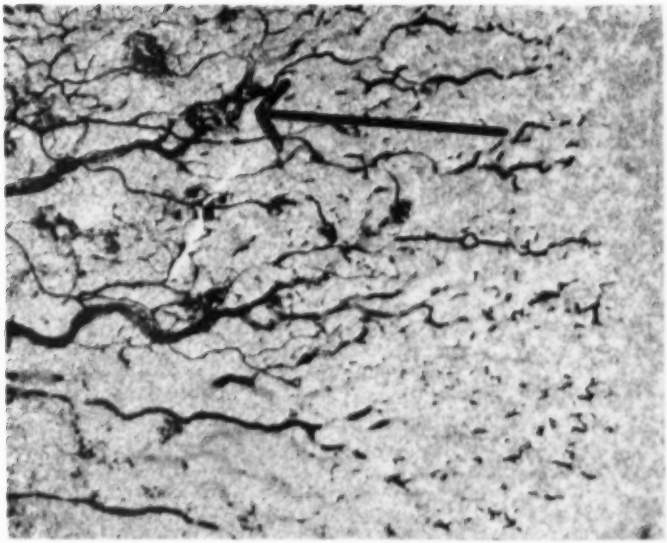
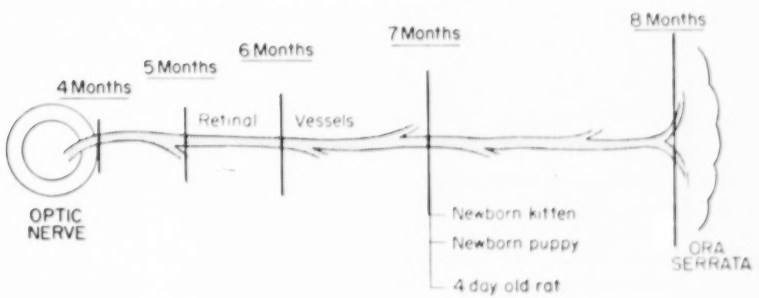


FIG. 8.—Flat retina preparation showing abnormal capillary tufts at the periphery of vascularization. Note avascular zone distal to retinal vessels. (26-day-old kitten, India ink injected. X50.)

The common factor fundamental to all these animal experiments is that the animal's eye is susceptible to oxygen only when the retina is incompletely vascularized and is resistant once vascularization is complete.

Figure 9 shows schematically the chronology of retinal vessel maturation in the human fetus based on the data of Mann and Michelson. The retinal vessels leave the disc at approximately four months gestation and reach



Schematic drawing showing chronology of retinal vascularization in human fetus. The retinal vessels advance from the Disc to the Ora between the 4th and 8th months of gestation. The relationship to retinal vessel development in the rat, cat, and dog are indicated.

FIGURE 9.

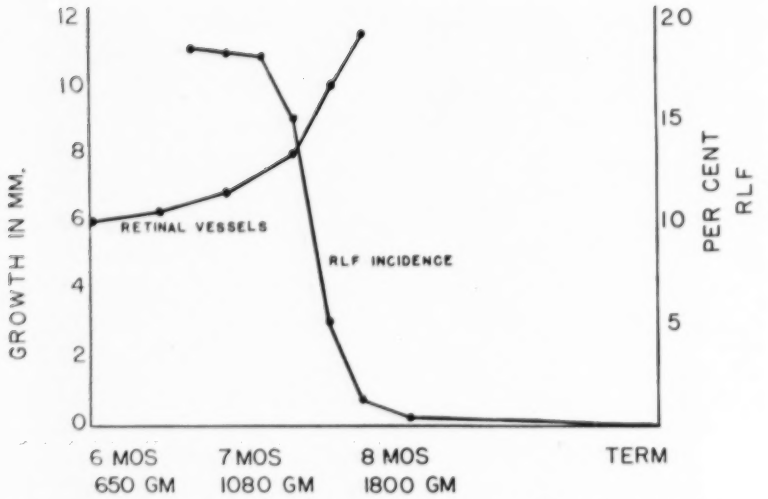


FIG. 10—This chart shows the correlation between incidence of retrolental fibroplasia and degee of maturation of the retinal vessels based on gestational age. (Nursery data collected between July, 1948, and December, 1950.)

the ora serrata at approximately eight months. It is a fortunate circumstance that in the experimental animals studied, many full-term newborn animals have an incompletely vascularized retina. For example, the newborn kitten or puppy and the four-day-old rat retinas are vascularized to the same degree as a seven-month human fetus.

Extensive examinations of all other organs in mice, rats, and kittens have failed to reveal changes produced by oxygen in other organs, except for occasional pulmonary edema on prolonged exposure.

The retinal response to oxygen in the animals studied in our laboratory is not only dependent upon an immature retinal vasculature, but is inversely proportional to the degree of vascularization of the retina (fig. 10). On the basis of these animal observations, the incidence of RLF in our premature nursery was reviewed on the basis of gestational age as an index to maturation of the retinal vessels.

A strikingly similar correlation between growth of retinal vessels and RLF incidence was noted and gives additional support to the evidence that the oxygen induced experimental lesions are probably analogous to early human RLF.

Dr. Friedenwald[5] has already pointed out that in human RLF, the abnormal capillaries are surrounded by a mucopolysaccharide substance stainable with Schiff or Hotchkiss stain, whereas in normal infants the capillaries do not take this stain. The histochemical similarity between the human and experimental lesions which react alike adds further evidence to the identity of the two lesions.

One inconsistency in the identity of the experimental and human lesions is the relative infrequency of retinal detachment in the animals exposed to oxygen. In our own laboratory the characteristic early proliferative changes of retrolental fibroplasia have been produced in several hundred animals. Most

of these have included mice and rats where detachment might not be expected as the lens, by its relatively large size, would tend to support the retina in its normal position. In only two puppies of twenty studied has detachment of the retina resulted from oxygen exposure. Here extensive hemorrhage into the vitreous preceded the retinal detachment. In approximately one hundred kittens exposed to an increased oxygen environment, retinal detachment was not observed.

The effects of oxygen on the incompletely vascularized retina can be divided into two phases: (1) an initial vasoconstrictive and obliterative phase and (2) a secondary proliferative phase. Several experiments have been performed in an attempt to further elucidate the mechanism of action of oxygen on the immature retina. Some of these are briefly cited.

1. *Sympathectomy.* The right vagosympathetic trunk was sectioned in four newborn puppies. These were placed in oxygen at 70 per cent concentration continuously. There was no difference between the right sympathectomized eye and normal left eye in the degree of vasoconstriction or obliteration in the retina in animals sacrificed at two days and five days; there was likewise no difference between right and left eye in degree and pattern of abnormal secondary vasoproliferation in the 15-day-old and 21-day-old puppies.

2. *Vasodilators.* The addition of 5 per cent carbon dioxide, a powerful vasodilator, combined with oxygen did not influence the initial vasoconstrictive or secondary proliferative effect of oxygen in mice and rats. Nicotinic acid, another dilator, did not alter the retinal vasoconstriction of oxygen.

3. *Retinal maturation during oxygen inhalation.* Normal vascularization of the immature animal retina is suppressed in most instances during high oxygen administration. Special staining techniques show that the nonvascular elements, however, are not visibly affected and differentiation continues. As a result, the normal channels of vascularization are possibly filled in by other elements during oxygen exposure. When vascularization is ultimately activated, the new vessels, lacking their normal channels, apparently erupt through the internal limiting membrane into the vitreous.

4. *Concentration of oxygen.* In the rat, oxygen concentrations under 40 per cent do not appreciably constrict or obliterate the retinal vessels at any age. From birth to 4 days of age, 40 to 55 per cent oxygen causes advanced vasoconstriction with minimal vasoconstriction in animals 4 to 8 days of age.

Concentrations of 60 to 70 per cent produce partial to complete obliteration of retinal vessels in all tested animals under 5 days of age, which is more pronounced in younger animals (fig. 11).

These data suggest, within the limits imposed by the differences in newborn animals and premature infants, that concentrations of oxygen under 40 per cent may be reasonably safe in the premature nursery. The data also suggest that the more premature the birth, the more susceptible is the infant to a given concentration of oxygen.

5. *Experiments on anoxia.* In view of the possible role of *simple anoxia* in RLF several experiments were performed. Newborn mice and rats were raised to 21 days of age in oxygen concentrations measuring between 10 and 14 per cent. The mortality rate at these low oxygen tensions was approximately 50 per cent. In those surviving animals the capillary-free zone around the retinal arterioles was slightly narrowed,

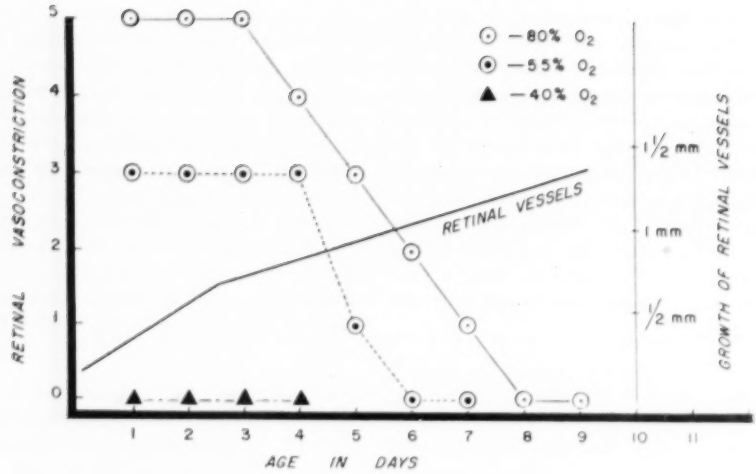


FIG. 11—Susceptibility to oxygen at various concentrations in the rat. Note correlation with maturation of retinal vessels.

but the vessels were perfectly normal in cross section; no endothelial nodules were noted, and in no instance did the vessels erupt from the retina into the vitreous.

In their preliminary report Ashton and co-workers[1] had suggested that exposure to a lowered oxygen environment might produce RLF lesions experimentally. On extending their experiments, however, they have reported[2] that retinal vessel proliferation into the vitreous did *not* occur on exposure to low oxygen tensions. These experimental data from both Ashton's and our own laboratory indicate that a *lowered* oxygen environment, even at levels where many premature infants would probably not survive, is inadequate to produce the classic RLF lesions experimentally.

It can be concluded that the marked vasoconstriction produced by oxygen in the immature retinal vessels is markedly exaggerated when compared to the slight constrictive effect on mature retinal vessels, or on vessels in other organs. Where the oxygen exposure is of short duration, the vasoconstriction is reversible on removal to room air. Where the oxygen exposure lasts for three to four days or longer, the vasoconstriction is irreversible and a true obliteration of the vessels results. Ashton and co-workers[1] were the first to differentiate the simple vasoconstriction from temporary exposure to oxygen from the true obliteration after a prolonged exposure to oxygen, an observation which has been amply confirmed in our own laboratory. Their observations with anticoagulants suggest that the obliteration results from an adhesion of opposing endothelium of the vessels or from intravascular clotting.[2]

The experimental production of lesions closely resembling RLF in animals raises many questions concerning the effects of oxygen on the immature retina. Working hypotheses[2,12,13] have been ventured to explain many of the observed phenomena, but these are beyond the scope of this report. The striking similarities between the experimen-

tal and human lesions should permit us to apply, with a reasonable degree of confidence, information gained from animal experimentation to problems in human RLF.

SUMMARY

I. The following qualities of the oxygen-induced experimental lesions justify the conclusion that these are the animal counterpart of early human retrolental fibroplasia:

A. Similar ophthalmoscopic changes in the experimental animals progressing from a preliminary phase of vasoconstriction through the early ophthalmoscopic stages of human RLF.

B. The experimental lesions are identical in morphology to the following that are observed microscopically in the human disease:
   1. Endothelial nodules in the nerve fiber layer of retina.
   2. Proliferation of retinal capillaries through the internal limiting membrane into the vitreous.
   3. Formation of glomerular-like capillary tufts.
   4. Vitreous disorganization.
   5. Retinal and pre-retinal hemorrhages.
   6. Retinal edema.

C. Identical histochemical response to PAS Schiff stain of experimental and human lesions.

D. The response of the immature animal eye to oxygen is inversely proportional to the degree of vascularization of the retina. This relationship correlates closely with the pattern of susceptibility of the human retina where RLF susceptibility varies inversely with degree of maturation of retinal vessels.

E. In human RLF, the eyes alone are usually involved without changes in other organs. In experimental animals exposed to a high oxygen environment, the lesions are limited to the eyes.

II. The experimental observations support the clinical impression that oxygen administration is intimately associated with RLF development.

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Addendum

Gerschman and co-workers (Am. J. Physiol., 179:115-118 [Oct.] 1954) have reported on independent studies on the effect of oxygen administration on the eyes of new-born mice. The lesions described conform closely to those herein illustrated.

Symposium: Retrolental Fibroplasia (Retinopathy of Prematurity)

MANAGEMENT

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IOWA CITY, IOWA

THE management of this disease is still only a palliative one. However, pediatricians and ophthalmologists should be acquainted with the various problems that may arise during the course of this condition. They may not always be able to cope with them, but they may frequently alleviate them. They should be able to reassure the parents, to counsel them as to prognosis and final outcome of the disease, and to advise them as to further educational aid and social adjustment in the case of severe visual impairment of the child.

THE ACTIVE PHASE

In order to detect active cases of retrolental fibroplasia premature children have to be examined as early in life as possible. It is usually feasible to examine a baby during the first week of life. In order to detect the exact onset of the disease and follow its development closely the infant should be seen once a week.

Good dilatation of the pupils is absolutely necessary for a satisfactory examination of the fundi. One drop of a 2 per cent homatropine hydrobromide solution and one drop of a 10 per cent phenylephrine (Neo-synephrine®) hydrochloride suspension are instilled into each eye. The phenylephrine occasionally causes blanching of the conjunctiva and lids in very small babies. The dilatation of the pupils is adequate after an hour even if the iris is heavily pigmented. Only in a very few affected children is dilatation unsatisfactory after these mydriatics. Atropine should be avoided in small children.

For the examination itself, the baby is taken out of the incubator, put on a table, and immobilized. This is easily achieved by wrapping the child in a blanket. Ophthalmoscopic examination is possible with the infant in an incubator, but is thus done only when the condition of the baby is precarious. The examiner stands at the baby's head and opens the lids with his fingers or instructs a nurse to pull down the lower lid as he raises the upper lid. The nurse stands at the infant's feet and grips the head firmly. In this way a satisfactory examination in the large majority of smaller infants is possible. Only larger or restless babies need to be pacified with a moistened, sterile sugar teat.* The sugar teat is generally effective in placating the baby up to the age of four months, after which he is less tractable and it becomes necessary to use a general anesthetic in indicated cases.

The fundus of a small premature child may in the beginning be obscured by a vitreous haze or clouding. Only some of the vessels may be faintly visible. The disc is often greyish or whitish

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* About one-third of a teaspoon of cane sugar is put in the middle of a sterile 2 x 2 inch sponge and then tied with a piece of thread.

in color. It may be best visible with a high myopic lens. As the eye matures, or in larger premature children, the vitreous clears and only remnants of the hyaloid system remain present. These also disappear quickly. The extreme periphery of the fundus has a characteristic, homogeneous grey color and appears elevated. This greyish peripheral zone persists for a considerable period of time and should not be confused with the peripheral changes in early retrolental fibroplasia.

The ophthalmoscopic examination should not be confined to the posterior segment, but should include the periphery, where the first pathologic changes may appear. It is usually possible to rotate the child's eye in any direction desired by exerting gentle pressure with the fingers holding the lids.

Infants with normal fundi are not followed after the age of three months. When the patient is discharged before that age, ophthalmoscopic examinations are made every two weeks in a follow-up clinic, the same technic being employed.

If retrolental fibroplasia is suspected or detected during one of these routine examinations, the baby should be followed more closely. It may be necessary to examine the child twice or three times a week in order to appreciate various progressions or regressions during the course of the disease. Only when the disease has progressed to a more severe stage should the parents be informed and instructed. It means unnecessary anxiety and worry for the whole family if they are informed about the child's condition at an earlier stage of the disease when the chances of a spontaneous regression are so great.

In the severe stages of the active phase the pupil should be intermittently or continuously dilated to avoid posterior synechias.

THE CICATRICIAL PHASE

When the active phase is over and the disease has run its course, the management of the case will depend upon the degree of severity into which the disease has developed. The prognosis for the milder cases is excellent and we can only assure the parents that the condition will usually remain stationary.

Severe cases should be followed at regular intervals. This should be every six months the first two years and then yearly. These later examinations serve mainly as a check on the development of possible complications. They also give the parents an opportunity to discuss with the physician any problems and difficulties which have come up during the preceding year.

Medical treatment of these severe cicatricial stages is of no avail. Surgery, irradiation and other therapeutic methods have been tried, but all these attempts have proven unsuccessful.

The remaining vision will depend on the severity of the case. Eyes affected with retrolental fibroplasia of Grade IV or V will have only light perception. In Grade III vision is 3/200 to 10/200 when the fold is on the temporal side. It may be better when the fold does not cross the macular area. In these severe cases fixation will develop later than normally and a roaming nystagmus may persist for a long period of time.

COMPLICATIONS

The most frequent complication of retrolental fibroplasia is secondary glaucoma. It develops in a fourth to a third of all severe cases. It is probably caused by an obstruction to the outflow of aqueous when the anterior chamber becomes increasingly shallow. Frequently the rise in intraocular pressure is brief and does not cause much discomfort to the child. Occasionally, however, the rise in

FIG. 1—Peripheral corneal opacities caused by anterior synechias.

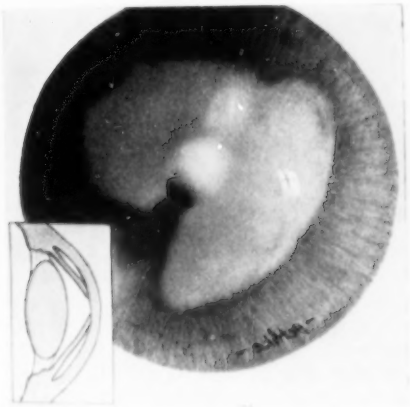

FIG. 2—Central corneal opacities caused by anterior synechias.

pressure may be acute and cause severe distress to the infant.

Glaucoma usually arises in eyes which are blind, and therefore no treatment is indicated except when pain and other general symptoms occur. This type of glaucoma may be controlled by miotics and the pressure will be normal after a few days or weeks. In a few cases it may be necessary to perform an operation to relieve the glaucoma if medical treatment alone does not suffice. However, the opening of an eyeball in such a patient may be followed by disastrous complications.

Few of the glaucomatous eyes in these children become buphthalmic as the glaucoma is usually of a transitory nature. Shallowing of the anterior chamber during a period of increased intraocular pressure may be followed by a deepening of the anterior chamber when the eye becomes atrophic.

Another frequent complication of severe retrolental fibroplasia is a corneal opacification. It is caused by anterior synechias and may present a cosmetic problem (figs. 1 and 2).

Another cosmetic problem is the smallness of the eye, deep set in the orbits, as enophthalmos occurs often in severely affected eyes (figs. 3 and 4). If the eye is completely blind it can be managed like any other atrophic eye.

A retinal fold (partial retinal detachment) that is seen in the cicatricial phase of the disease usually does not progress. Such an eye may deviate, thus enabling the patient to use an attached part of the retina for fixation.

Esotropia of the more amblyopic eye is a common complication. It can be surgically corrected for cosmetic reasons. This should not be done when the deviation brings a functioning area of the retina into the line of fixation.

Cataract develops only in about 3 per cent of eyes affected with Stage IV or V. It is therefore an uncommon complication of this disease.

EDUCATIONAL AND SOCIAL ADJUSTMENT

As long as no effective treatment for retrolental fibroplasia is available these patients will present an educational and social problem more than a medical

FIG. 3—Deep-set eyes in severe retrolental fibroplasia.

problem. The parents have to be guided in their endeavor to bring up a visually handicapped child. Many agencies and trained social workers will be able to give help to them, but the first responsibility lies with the physician who makes the diagnosis and informs the parents about the poor prognosis.

While the physician has to discuss with the parents many medical problems of retrolental fibroplasia, he will have to refer them to the proper agency for further counseling. The panel has prepared a pamphlet written for parents of children with retrolental fibroplasia. This booklet is to be distributed by the physician and it should bridge the gap between the physician and the social worker. It will enlighten the parents about many problems of the disease.

FIG. 4—Lateral view of deep-set eyes in severe retrolental fibroplasia.

CONCLUSIONS

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ALL theories concerned with the etiology of RLF excepting that concerned with oxygen have failed of confirmation. We feel that we can recommend with confidence a program entailing a more rational use of oxygen in the management of the premature infant and that we may anticipate a significant drop in the incidence of this disease, which has become the greatest cause of blindness in children.

While great advances have been made in the better understanding of the etiology of RLF, it is probable that even with judicious use of oxygen there will be occasional cases of RLF. It is hoped that future investigations will reveal other etiologic factors.

It must be remembered, however, that use of oxygen is sometimes a lifesaving measure, and it will be necessary to continue its use for infants with cyanosis and definite signs of respiratory distress.

Pediatricians throughout the country are taking the facts into consideration in the mangement of newly born prematures. The indications for oxygen administration are now considered to be fewer than they have been in the recent past. Cyanosis and respiratory distress are held to be bona fide symptoms requiring oxygen therapy. However, there is a growing conviction that irregular or periodic respirations unaccompanied by other signs of distress do not require oxygen administration. When oxygen is indicated, every effort is made to keep the concentration in the incubator below 40 per cent and to discontinue treatment as soon as respiratory distress is relieved.

We feel that there is insufficient evidence available to support the idea that RLF is benefited by oxygen therapy.

The panel feels also that there is insufficient evidence available to warrant a statement that the rate of withdrawal affects the ultimate outcome of the disease. Controlled animal experimentation should help clarify this important issue.

The remarkable similarity between the oxygen-induced experimental lesions and the lesions of early human RLF suggest that the true counterpart of human RLF has been produced. Because of the close identity of the animal and early human lesions, data obtained from animal experiments can be applied with reasonable confidence to problems in human RLF. One inconsistency, however, is the infrequency of retinal detachment in the oxygen-treated animals. The data presented show that the common denominator in all the animal experiments is a susceptibility to oxygen where the retina is incompletely vascularized and a resistance to oxygen damage where vascularization is complete.

DISCUSSION

QUESTION: *How do you correlate stage I of vasodilatation with the vasospasm of the experimental animal?*

DR. PATZ: I think the vasospasm seen in experimental animals can be correlated to a certain extent with observations in the clinical disease. During the first year of our controlled nursery study, four of seven infants in oxygen at concentrations of 60 to 70 per cent showed marked vasoconstriction and obliteration prior to the appearance of vasodilatation. This was a casual observation and we were unaware of its real significance.

During the second year of the study, we looked particularly for this change, and all five cases in high oxygen that developed terminal membranes progressed through an initial stage of marked attenuation or obliteration of their retinal vessels prior to dilatation.

It is important to point out that these infants received oxygen at 60 to 70 per cent concentrations. I think probably at lower oxygen concentrations the marked attenuation and obliteration of the vessels, particularly where they were visible in the posterior segment, would not occur, although the more peripheral complexes would show vasospasm.

On one of the animal slides I have just shown, the posterior segment appeared relatively normal with the abnormal capillary tufts limited to the periphery.

QUESTION: *How do you explain retrolental fibroplasia developing in full-term infants on your scheme of retinal vessel growth?*

DR. PATZ: It seems reasonable, on the basis of our animal studies, that considerable variation in the rate of growth of the retinal vessels could occur, and I can conceive of a full-term infant whose vessels had just reached the ora serrata but in whom vascularization had not been completed.

In animals there is considerable variation in retinal vessel outgrowth for a given age, and we noticed this much more in our kittens than we did in the white rats. In rats we were dealing with a pure strain of animals with a common genetic background, whereas the kittens were offspring of ordinary alley cats, and I would question their genetic background. I think a reasonable degree of variation in human retinal vessel maturation can also be anticipated.

QUESTION: *How do you explain those retrolental fibroplasia cases which get no oxygen?*

DR. PATZ: I would prefer to explain those on mechanisms other than oxygen, simply adhering to the hypothesis that oxygen is an important, and possibly the principal factor in the development of the disease but that other factors may be basically related. It can be explained within the framework of the oxygen theory, however, on the basis that the fetus in utero has a lowered arterial oxygen content with hemoglobin saturation approximating 50 per cent. After birth these premature infants, even in room atmosphere, show a rapid rise in arterial oxygen to approximately 90 per cent saturation. It is reasonable to postulate that this increase from the relatively low tensions sustained in utero might account for the very rare case that occurs without supplementary oxygen administration.

DR. FRIEDENWALD: I should like to make some general comments about the questions as a whole, as I have seen them arriving at the desk. Apparently the questioners hold the panel in very high esteem and expect the panel members to answer all the questions that could possibly be formulated about this disease. In fact, I believe we are expected, so to speak, to have a full-dress theory of this disease, complete with white tie and tails, available for you. That is a very nice thing for you to think. However, I believe that there is no disease about which a full and complete theory is at present available, so we need not feel too badly over the fact that there are many of these questions we cannot answer.

Among questions that were directed to me are several about the orbital changes that are alleged to be connected with this disease.

QUESTION: *How does one explain orbital edema in the active stages of the disease?*

DR. FRIEDENWALD: My own feeling is that orbital edema is not a regular or necessary factor in the disease, and I certainly have no explanation for it if it exists.

The second question about the orbit asks whether the apparent enophthalmos that has been demonstrated in several pictures indicates orbital atrophy or not. My own feeling is that it does not indicate orbital change, but merely the fact that these eyes are microphthalmic and that the enophthalmos is apparent rather than real.

QUESTION: *Have these following factors been considered in the evaluation of oxygen therapy? Continuously recorded oxygen analyses of incubators have demonstrated three things*: (1) *marked variation of maximum and average concentration and time at or above the average concentration;* (2) *variation of concentration between types of incubators and settings of the same types of incubators, and* (3) *unexplained variations of oxygen concentration.*

DR. KINSEY: We are certainly cognizant of all these factors, and we tried our best to take these variables into account by taking repeated readings of oxygen concentration and then averaging the values obtained. However, this method only partially resolves some of the problems of interpreting the results and, therefore, too much weight should not be placed on the average concentrations reported. I think one can only generalize with regard to concentration.

QUESTION: *Can ocular damage be produced by placing newborns in oxygen-mixed atmosphere by means of a funnel?*

DR. KINSEY: Presumably yes, if the time of exposure is long enough. Cases of retrolental fibroplasia have been reported from Cincinnati following the administration of oxygen by means of a funnel.

QUESTION: *If rapid transfer to normal air is followed by changes indicating retrolental fibroplasia, what is the rationale of condemning gradual weaning?*

DR. KINSEY: The rationale for condemning gradual weaning is based on the evidence presented, which shows that if you place a baby in oxygen four days instead of two days, the incidence rate doubles. Thus prolonging the stay in oxygen increases the risk of contracting retrolental fibroplasia. To balance this risk it would be necessary to have better evidence than I think is now available that rapid withdrawal increases the incidence of retrolental fibroplasia to a greater extent than the extra exposure to oxygen which may be involved in gradual weaning.

DR. BLODI: I have a number of questions regarding oxygen therapy and withdrawal rate. DR. REESE has answered these questions in a more or less general way in his closing remarks and I have nothing to add.

QUESTION: *Why do these children rub their eyes with their fists?*

DR. BLODI: I think there are two explanations. One is that this gives them a mechanical stimulation of the retina and they enjoy seeing light stars and other visual images. The other explanation is that such behavior is psychologic.

QUESTION: *How does one prevent or manage intraocular hemorrhage in the cicatricial stage?*

DR. BLODI: I don't know of any way of preventing it, and the treatment chosen depends very much upon the intraocular pressure.

QUESTION: *What is the frequency of myopia as a residual?*

DR. BLODI: I think it is quite high, and I believe that DR. REESE in his forthcoming paper will give the exact figures on that.

QUESTION: *Is there any evidence of an identical pathogenesis and etiology with hemangioblastoma?*

DR. SILVERMAN: We have only clinical observations to report, although the histologic appearance of the cutaneous hemangioma that occurs in premature infants resembles that of retrolental fibroplasia very closely. Experience in our own nursery indicates that there is a relationship between the incidence of postnatally appearing hemangiomas and the degree of prematurity. Among the more immature infants there is a higher incidence of hemangiomas. However, there is no dependent relationship with retrolental fibroplasia.

I should like to mention a recent observation which, I must warn, is at this time merely a tenuous suggestion. We have been plotting the incidence of postnatally appearing hemangiomas for the past five years, and we have observed that the incidence of these skin lesions decreased when the prolonged administration of oxygen in high percentage was discontinued as routine procedure. Whether this decrease was real or merely the result of chance must await a larger experience. I mention our experience to stimulate others to look for a similar association.

QUESTION: *Two variables in the incubator environment of the prematures, moisture and heat, have not been discussed. Do these two factors have any significance in the etiology of retrolental fibroplasia?*

DR. SILVERMAN: I think this question can be answered by saying that there is no published evidence that either of these factors is important in the production of the disease. There is no reason to suspect that moisture would be involved. It is possible that heat is a factor, and perhaps the role of heat will be further elucidated in animal work. There is no clinical evidence or published experimental evidence to bear on this point.

FAMILIAL PRIMARY HYPOPLASIA OF THE ORBITAL MARGIN

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"All cases are unique, and very similar to others." (T. S. Eliot, *The Cocktail Party.*)

ALTHOUGH the ever-growing number of developmental abnormalities of the skull and the face registered in literature makes their classification difficult, and though, as pointed out by Duke-Elder,[9] the tendency has been to accept as separate nosological entities what appear merely to be the result of a variable anatomic localization of the same fundamental disturbance, it seems hardly possible to deny the existence of several distinct, basic types of such aberrations, which possess definite, clearly distinguishable morphological features, respond to different pathogenetic mechanisms and are loaded with entirely disparate hereditary tendencies.

With a purely descriptive and topographic criterion, Vogt[42] divided those developmental dysostoses affecting the head into the two main categories of *dyscrania* and *dysprosopia,* according to whether the exclusive or predominant site of the defect is the cranium or the skeleton of the face; cases in which the head is involved as a whole were given by him the name of *dyscephalia.* More recently, Nager and deReynier[25] adopted the same point of view and endeavored to classify the majority of the previously described craniofacial malformations according to it and to whether or not other deformities were present at the level of the extremities. By adhering to their line of thought, the following groups may thus be established:

1. The *dyscranias,** in which the neurocranium is involved, either alone or in association with developmental errors of the hands and feet, as in Apert's acrocraniosyndactyly. Most types entering Virchow's original arrangement of craniostenoses,[41] and most comprised in Brown and Harper's, Simmons and Peyton's, and Fairman and Horrax' classifications (oxycephaly, scaphocephaly, brachycephaly, trigonocephaly, platicephaly and hypertelorism),[5,33,10,11] should be grouped under this heading, as should also Franceschetti's *dysostose cranienne avec calotte cérébriforme,*[12] since they are all supposed to depend on the obliteration of one or more cranial sutures.

The accompanying anomalies of the facial bones are then accounted for on mechanical grounds and must be viewed as secondary in nature.

Concurring in what may be thought by some to be an over-simplification, Nager and deReynier[25] have reduced all types of dyscrania to three basic forms: brachycephaly, dolichocephaly and Crouzon's skull, which would be conditioned, respectively, by the involvement of the coronal, the sagittal, and both the coronal and the lambdoid sutures. The fact that oxycephaly is not considered by them a distinct, separate clinical entity might be due to their not

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*As, linguistically, the term "dyscephalia" used by Nager and deReynier for referring to this group is utterly inappropriate in that it implies the involvement of the whole head, I have preferred to substitute for it the more suitable term "dyscrania." The denominations of dyscephalia and dysprosopia given to the second and third groups, respectively, were not used by these authors and have been taken by me from Vogt's publication.

having been faced directly with such malformation. Fairman and Horrax' decision[10,11] to eliminate Crouzon's disease from their classification—in which, on the other hand, Apert's acrocraniosyndactyly is not even mentioned—on account of Crouzon's description not being clear enough, rests probably on similar grounds.

2. The *dyscephalias,* in which the whole skull, or at least part of the vault or the basis and part of the osseous scaffolding of the face, is involved, either alone (Crouzon's craniofacial dysostosis) or in association with defects of the distal end of the limbs (Vogt's dyscephalodactyly).

3. The *dysprosopias,* in which the dysontogenetic process interferes with the development of the facial bones and which comprise all conditions pertaining to what has been called by François[16] the *first branchial arch syndrome.* Franceschetti and Zwahlen's mandibulofacial dysostosis,[15] Hallermann[18] and Streiff's special form of mandibulofacial dysostosis,[35] Villaret and Desoille's familial primary hypoplasia of the upper maxilla,[40] and Nager and deReynier's mandibular dysostosis,[25] are to be assembled at this place, as is a wide range of malformations involving the mandible (hypoplasia of the lower jaw, which in extreme cases, as in otocephaly, may be entirely absent), the external ear (aplasia of the pinna, atresia of the external auditory canal, pre-auricular appendages, fistula auris congenita) and even the eye (epibulbar dermoid and lipodermoid tumors, microcornea, microphthalmos, congenital cataract, multiple colobomas).[17,20,25]

Franceschetti and Klein[14] feel that the combination of strictly facial disorders with anomalies of the extremities is purely incidental, the latter representing only an additional character, related to the palatoschisis with which these patients are frequently afflicted and not to the dysostosis itself. The name of acrofacial dysostosis proposed by Nager and deReynier for these cases, therefore, would hardly be justified.

Although intended at its inception to be sheerly empiric, this regional classification lies indeed on firm ontogenetic foundations, for while the bones of the vault and the base of the skull derive for the most part from the paraxial mesoderm, it is the visceral mesoderm coming from the paired first branchial arches which goes to make up the osseous support of the face. Besides, it must be noted that although the premature closure of one or more sutures seems to play a major role in the causation of deformities of the calvarium and of the base of the skull, which accordingly deserve to be considered as true craniostenoses, it is an atypical development of the bones themselves, i.e., a developmental defect in their orderly growth, which, as evidence strongly suggests, is at the bottom of all malformations involving the face.[14] In passing, however, it could be remarked that the obliteration of the cranial sutures may well be the result, and not the cause, of inhibited growth of the interstitial mesenchyma which normally separates the skull bones[28] and which would fail to respond to the stimulus represented by the growth of the brain.[22]

The observation of a hitherto undescribed form of craniofacial aberration which consists chiefly of a marked hypoplasia of the orbital margin, a severe maldevelopment of the tarsal plates and a congenital defect of the lacrimal passages, and which possesses a strong hereditary tendency, has led me to report its main features and to present the pedigrees of two identically affected families.

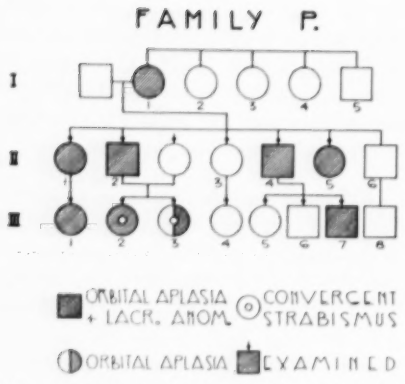

FIG. 1.—Genealogic chart showing dominant mode of transmission of primary hypoplasia of the orbital margin in a family of three generations.

FIRST FAMILY

The first member of this family (M.P., fig. 1, III, 2; fig. 2), now a ten-year-old girl, was referred to me in 1947, at the age of four, because of a periodic convergent strabismus which had been noticed for the past few weeks. Both parents were fully aware of the queer appearance of the girl's face and of the existence of some abnormality about her lacrimal passages, but no special significance was attached to them, in spite of the existence of tearing, as they were known to be of common occurrence in the paternal lineage and were present in the father himself. The girl was born at full term after an uncomplicated pregnancy and labor. Her physical and mental development had been normal, with no antecedents of headaches or convulsions. No consanguineous marriages had taken place among members of at least the two previous generations, and no history of other congenital defects or of miscarriages was elicited.

Physical examination. The patient was well developed and well nourished and appeared alert, interested and intelligent.

A striking facial deformity was noted at first glance: the margin of the orbit was flattened, as if in modelling the head in fresh clay an artist had depressed forcibly both eye sockets by placing the palms of his hands over them, each on one side of the root of the nose.*

This hypoplasia was as noticeable at the inner part of the orbital rim, where the internal canthal ligament stood out clearly under the skin, its tenseness and resiliency being conspicuous to the eye, as it was at its inferior and especially at its external part, which occupied a situation much posterior to that encountered normally. Likewise, the outer half of the superior margin was depressed to the point of having no relief whatsoever, and as the malar bones were also flattened, the base of the orbital pyramid sloped steeply outwards, forming a particularly acute angle with the orbital axis. As a result of this, the lids and the eyeballs themselves were seen, when observed from one side, to have an undue prominence.

Owing to the malformation, ordinary ophthalmometric readings could not be relied on; yet it was evident that no degree of proptosis really existed. As the palpebral apertures, which had a strictly horizontal course, were not enlarged, the eyes resembled those of a frog.

The zygomatic arch and the temporal region were markedly protuberant and the forehead appeared high and broad. A shallow sagittal furrow, 5 cm. in length, could be felt above the external occipital protuberance. The upper jaw was otherwise well developed, as were also the intermaxillary bone and the mandible. The teeth were normal and correctly aligned. She had a closed bite and a normal palate.

The auricles were large and of normal design, but stuck out from the side of the head. The external auditory canals and the tympanic membrane were normal. Functional examination failed to reveal any auditory deficit or vestibular disturbance.

The clavicles, scapulas and the bones of the thorax, arms, forearms, hands, legs and feet were all normal.

Ocular examination. On close inspection, the skin of the lids was found to be very thin and extensible, of a nearly atrophic quality; through it, many small, slightly prominent dermic veins were visible. Although the lashes were normal and had a regular distribution, the tarsal plates were thin and narrow, and almost as pliable as the neighboring soft parts.

The upper lacrimal puncti were present and normally placed; the lower ones, much displaced nasally, were represented by a narrow slit, 1 mm. long, which pierced the lid margin at a distance of about 1 mm. from the inner canthus. On the other hand, a supernumerary canaliculus opened immediately before the caruncle, behind the mucocutaneous junction, and ran independently into the lacrimal sac,

*This image, although conveniently modified to fit the present situation, has been taken from Crouzon's description of dysostosis craniofacialis.[6]

FIG. 2—Familial primary hypoplasia of the orbital margin (M.P., case III, 2, fig. 1).

as could be demonstrated by Schoute's test.[31] Exploration with a no. 2 probe revealed that while all these passages were patent, a complete blockage was present at the upper ostium of the nasolacrimal duct. Some degree of lacrimal stasis existed on both sides.

Apart from a partially accommodative, periodic, alternating esotropia of a variable degree (esotropia of a III+I type, Urrets-Zavalia[37]), a slight overaction of the right inferior oblique could be seen in levoversion. The fundi and the refractive media were normal. Retinoscopic findings under atropine cycloplegia were as follows: O.D., +4 sph., + 0.50 cyl., ax. 85°; O.S., +2.50 sph., + 1 cyl., ax. 95°. Corrected vision was 20/30 in each eye. The interpupillary distance amounted to 53 mm.

As soon as the required correction was used, the squint disappeared; yet, it recurred sometimes when the glasses were removed and until recently the cure could not be considered complete. At present, though with the screen test it is still possible to disclose the existence of a slight esophoria, binocular vision is constantly in vigor, with and without glasses.

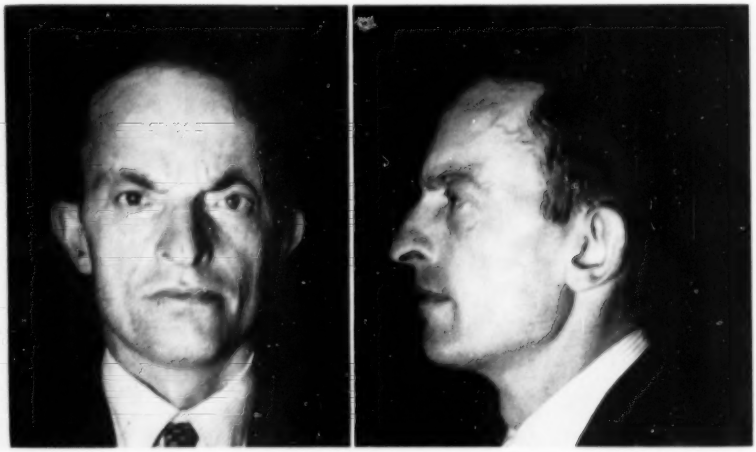

FIG. 3—Familial primary hypoplasia of the orbital margin (L.P., case II, 2, in fig. 1).

Laboratory findings. The blood and urine were normal and the serologic reactions negative.

Roentgenographic examination. The cranium exhibited a transversal enlargement but was otherwise of normal size; none the less, there were slight convolutional markings and an absence of the lambdoid suture line. The vault was rather thin. The sella had a normal appearance (fig. 2).

It is important to note that although the orbits were clearly underdeveloped in their anterior part, there was neither an abnormal brevity of the anterior fossa nor a depression of the orbital roof, which showed a gentle downward posterior slope as in normal individuals.

The radiologic sphenoidal angle, as defined by the three points designated as nasion, ephippion and basion, the nasal angle, as determined by the acanthion, the nasion and the basion, and that angle determined by the projections of the acanthion, the nasion and the ephippion which I should call the maxillary angle,[7,38] measured 121 degrees, 56 degrees, and 82 degrees, respectively. These abnormally low values revealed that a basilar kyphosis and an underdevelopment and recession of the orbital and malar regions existed. The lower part of the maxilla had, however, a normal development, as shown by the absence of the inferior prognathism which would otherwise have been present. The value of Cloquet's facial angle, which amounted to 72 degrees, revealed that on the whole the facial skeleton was small and gracile.

This girl's only sister (C.P., fig. 1, III, 3) was 19 months old when first seen in 1947. She was examined thoroughly because of the suspected familial character of the disease, and

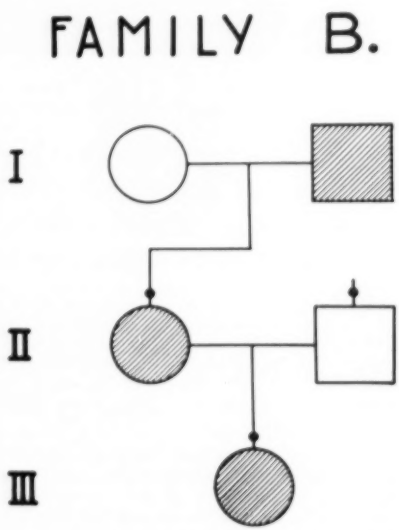

FIG. 4—Genealogic chart showing inheritance of familial primary hypoplasia of the orbital margin over three generations.

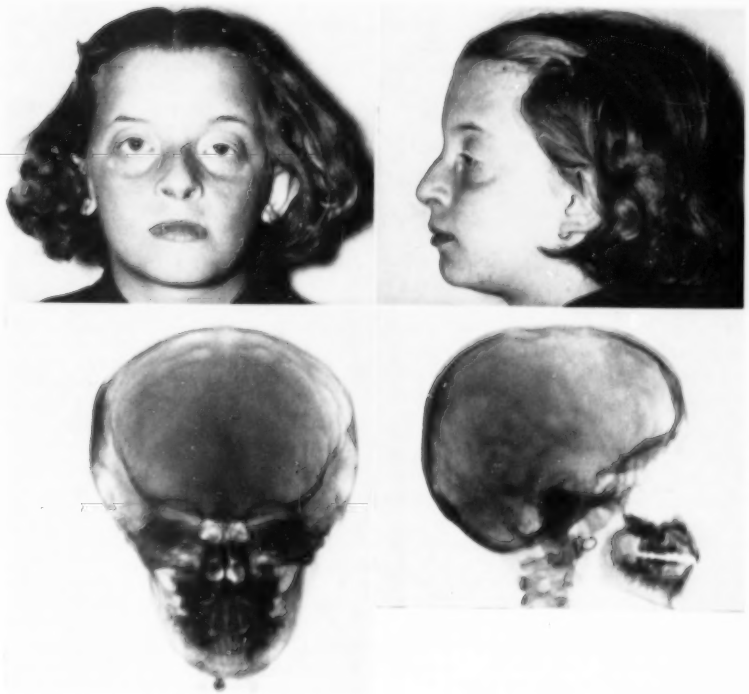

FIG. 5—Familial primary hypoplasia of the orbital margin (R.B., case III in fig. 4).

was found to exhibit the same peculiar features, though to a lesser degree. On close examination the palpebral skin looked very thin; through it, many fine dermic veins were seen to protrude slightly. However, no tarsal or lacrimal abnormalities were encountered. The pinnae and the external auditory meatuses were normal, as were also the dental arcades. The teeth were rather carious and frayed. There was no hypoacousia.

In 1951 she also developed a periodic alternating esotropia of a partially accommodative nature, which was kept under control with glasses but later required the assistance of orthoptic measures before the necessary disjunction between accommodation and convergence was reached. A slight but clearly visible overaction of the right inferior oblique was detected, as in her elder sister.

Under atropine cycloplegia, refraction was as follows: O.D., + 5.50 sph., + 0.75 cyl., ax. 90°; O.S., + 5.50 sph., + 0.50 cyl., ax. 105°. The media and fundi were normal. Corrected vision, as determined in 1953, was 20/50 in the right eye and 20/30 in the left eye. The interpupillary distance was 57 mm. at the age of six.

Radiologically, the skull was found to be of normal shape and dimensions. There were neither digital impressions nor a premature seclusion of the suture lines. The existence of a slight hypoplasia of the orbital and malar regions, plainly visible in lateral roentgenograms, was confirmed by craniometric measurements (nasal angle: 58 degrees; maxillary angle: 84 degrees). No basilar kyphosis was present, as in the former case (sphenoidal angle: 132 degrees).

The father (L.P., fig. 1, II, 2; fig. 3), a tall and strong farmer of 37, was unaware of any visual difficulty and said to have paid no attention to his facial deformity, which he had inherited from his mother. Being quite alert and cooperative, he lent himself willingly to examination.

The same cephalic disorder was present we found in his daughter M.P. Owing to the complete lack of salience of the external orbital margins, the face sloped steeply from the external canthi outwards, to the unduly prominent zygomatic arcades, without showing, as normally, a distinct palpable edge at the place of the former. The teeth had a regular distribution and were altogether normal, except in that the upper incisors were small and separated by intervals (*rake teeth*). He had a normal closed bite.

No other physical abnormalities were present. The vestibular and cochlear apparatus were normal on functional examination.

The palpebral skin and the tarsal plates were extremely hypoplastic. The upper puncta, being displaced nasally with respect to the corresponding papillae, opened on the lacrimal part of the lid margin; the lower ones were situated at the inner canthi, well behind the cutaneomucosal junction. There was no epiphora. On examination (syringing and probing), the lacrimal passages were found to be freely patent.

The media and fundi were normal. Refraction under homatropine cycloplegia was as follows: O.D., + 0.50 sph.; O.S., + 0.75 sph. Corrected vision amounted to 20/20 in each eye. No abnormality of the dissociated position was found, either at 20 feet or at 16 inches. Interpupillary distance was definitely though moderately increased: 70 mm.

Skull roentgenographs showed, aside from some slight convolutional markings, an enlargement of the bitemporal diameter. The suture lines were no longer visible. The orbits were underdeveloped. The upper jaw was otherwise normal, as was the lower one. The basal or sphenoidal, the nasal and the maxillary angles were only slightly reduced (128 degrees, 59 degrees and 84 degrees, respectively).

Subsequently, nine relatives of the father were examined, and five were found to present the same craniofacial deformity, the palpebral anomalies, a variable amount of hyperopia ranging from 1.5 to 5 diopters, and similar, if not always identical, lacrimal aberrations (fig. 1).

SECOND FAMILY

R.B., a girl of nine, was brought for examination in 1953 because of ocular discharge and tearing which had been present from birth (fig. 4, III; fig. 5). The mother was fully aware of the girl's rather strange look, but was flattered with what she considered a happy resemblance to her own features, which were also those of her father. The left eye was known to have always been "smaller" than its fellow. The past history of the patient was otherwise noncontributory. She was born at term, had walked and spoken at the normal age and was well developed and nourished. No consanguineous marriages had taken place in the family, which derived from Italian ancestors and was related in no way to family P., which was of Spanish origin. The patient was an only child, as were also her mother and her maternal grandfather.

On examination, I was surprised to note that, as in so many members of the first family, a distinct, isolated aplasia of the orbital margin was present, and even to a higher degree. The outer and lower borders were strongly underdeveloped and receding; the external two thirds of the upper border were very much flattened, and the superciliary ridges nonexistent. On the nasal side, the anterior lacrimal crest could not be made out by touch, and a hypoplasia, in the form of a depression, was felt at the level of the nasal process of the maxillary bone. The inner canthal ligament made a marked prominence under the skin.

As a result of all this, the base of the orbital pyramid showed a conspicuous slope toward the temples. The zygomatic arcades and the temporal regions were not abnormally prominent. The malar bones were very much flattened, but there was no aplasia of the lower part of the upper jaw or of the mandible. Although the intermaxillary bone was well developed and the dental arcades had a normal amplitude, the teeth were irregularly aligned. There was a closed bite. The palate had a normal curvature. The ears and the external auditory meatuses were found to be normal, as was also the labyrinth, on rough clinical examination. Although not very carefully investigated, the sense of smell seemed to be normal.

On close inspection, the skin of the lids appeared very thin; through it, turgid, small dermic veins were plainly visible. A slight degree of palpebral ptosis existed on the left, where the upper lid covered 4 mm., and not 2 mm. as on the right, of the corneal surface. No exophthalmos proper existed, in that the eyes were not abnormally prominent with respect to the bridge of the nose, the forehead or the lower part of the cheek, nor was the right palpebral aperture enlarged; however, as a result of the receding position of the orbital margin, the eyes and the lids protruded distinctly with respect to the latter and gave the patient, as in previous cases, a froglike appearance.

Some lacrimal stasis was present. The lower canaliculi and puncta were absent, the lower

lid margins showing in what should have been their lacrimal section a slight, almost colobomatous indentation, as if the part which normally contains the lower canaliculus were lacking. The upper puncta and canaliculi were normal and seemingly patent, as were also the nasolacrimal ducts, since florescein instilled into the lower fornices appeared at once in the nasal secretions.

The media and fundi were normal. Orthophoria was found at all distances. Refraction under atropine cycloplegia could not be determined, but direct, paramacular retinoscopy revealed that both eyes were nearly emmetropic. Uncorrected vision was: O.D., 20/30; O.S., 20/20. Interpupillary distance amounted to 55 mm.

Roentgenographic findings (fig. 5) were as follows: The skull was normally formed, had normal dimensions, and showed no convolutional markings; the suture lines were still plainly visible; the sella was normal. The roof of the orbit had no abnormal slope. A marked hypoplasia of the orbital and malar regions was clearly visible; the upper and the lower jaws exhibited a normal development, as shown by the fact that no inferior prognathism existed and that, although the nasal and the maxillary angles were moderately reduced (58 degrees and 82 degrees, respectively), the facial angle of Cloquet had a normal value (66 degrees). The basal angle measured 133 degrees.

The mother (R. de B., fig. 4, II), a woman of forty-six, resented openly the interest aroused by her features and lent herself reluctantly to examination. For that reason, neither x-rays of the skull nor photographs could be taken of her, nor cycloplegia of any sort performed on her or her daughter. Vision with the left eye had always been poor.

An obvious agenesia of the orbital rim, exactly alike to that shown by her daughter, was present, as were also the aplasia of the skin of the lids and of the tarsal plates and the aberration of the lacrimal passages. No other deformity of the vault or the base of the skull could be seen. The lacrimal passages could not be tested for patency, and all that may be said in that respect is that a conspicuous epiphora existed. The small colobomatous defect which occupied the inner sixth of the lower lid margin was more marked than it was in her daughter.

A slight left palpebral ptosis and 15 diopters of left esotropia were present. The media and fundi were normal. Direct, paramacular retinoscopic findings were as follows: O.D., + 0.50 cyl., ax. 85°; O.S., −3 sph., + 5 cyl., ax. 5°. Corrected vision amounted to 20/25 in the right eye and to only 20/200 in the amblyopic left eye.

Although the patient volunteered no information whatsoever concerning her family, it could be ascertained beyond any doubt that her father had presented the same craniofacial disorder and that he had always been known to suffer from a lacrimal defect and to have had some tearing and conjunctival discharge.

The sum of the collected material confronts us with a distinct and typical craniofacial deformity occurring through the overwhelming pressure of hereditary factors. The main constitutive elements of this symptom complex are:

1. A considerable hypoplasia of the orbital margin, which results from an underdevelopment of the following structures: the orbital arch, the internal and, principally, the external angular processes of the frontal bone; the frontosphenoidal and the marginal processes of the malar bone; and the foremost part of the orbital plate and the nasal process of the superior maxilla. No aplasia of the mandible or of the alveolar process and the body of the upper maxilla could be demonstrated.

2. A marked hypoplasia of the palpebral skin and of the tarsal plates.

3. A variable developmental defect of the lacrimal passages, which consisted in one family in an abnormal brevity of the inferior canaliculus, in ectopia and elongation of the lower punctum, in atresia of the nasolacrimal duct, and, in one case (fig. 1, III, 2), in a supernumerary canaliculus which opened at the inner canthus just before the caruncle. In the other family, a total absence of the lower canaliculus was seen, in conjunction with a shallow, scarcely noticeable colobomatous indentation of the lacrimal part of the lower lid border.

As in the case of other cephalic mal-

formations, a most manifest "air de famille" was apparent in all patients.

In two sisters belonging to the first family (fig. 1, III, 2, and III, 3), a left superior rectus insufficiency was detected, in association with a partly accommodative esotropia and with a moderate amount of hyperopia, which was also encountered in the remaining affected members of that family. On the other hand, both the patients of the second family who were examined, a mother and her daughter (fig. 4, II and III), exhibited a slight left palpebral ptosis which, in the former, was linked to a homolateral mixed astigmatism of a high degree.

In the second family, x-ray examination of the head revealed an agenesia of the orbital and malar regions as the only patent disturbance. In the first one, in addition, it showed evidence of more widespread defects involving the vault and the base of the skull, such as cerebriform convolutional markings, obliteration of the lambdoid suture line and a definite increase of the bitemporal diameter which, on the whole, corresponded closely with the deformity seen by Franceschetti[12] in his cases of cranial dysostosis with cerebriform calotte (pseudo-Crouzon).

Craniometric measurements regularly evinced a reduction of the maxillary and nasal angles consistent with the skeletal disturbances; furthermore, in the first family a slight to moderate basilar kyphosis was eventually present, as indicated by the relatively low value of the sphenoidal angle.

Apart from the grandmother, who was said to have transmitted the disease to her descendants, the first family comprised fifteen persons in the second and third generations, eight of which were affected; of these, seven presented the complete syndrome and one was found to suffer from the osseous and cutaneous malformations but not from the tarsal and lacrimal ones. In the second family, the grandfather, the mother and the daughter were fully afflicted.

Recently, the whole syndrome was observed by me in a case of marked acrobrachycephaly occurring fortuitously in a single member of a family.

J.C.S. (fig. 6), a twenty-three-year-old man, was known to have presented from birth a cranio-facial deformity and to have had lacrimation since the age of three months. No other relevant facts were found in his personal or family history. The eyesight and hearing had always been normal.

On examination, the patient was found to be normal mentally and physically, except for the notably distorted shape of the head. There were acrocephaly, brachycephaly and a marked hypoplasia of the malar region and of the orbital margin; yet the forehead presented short and extremely prominent, almost simian, supercilliary ridges in the glabella, which met in the midline. The root of the nose was very much depressed with respect to the latter. The auricles were of normal shape but stuck out from the side of the head; the mastoid regions were unduly salient, as were the zygomatic arcades. The teeth were normal and normally implanted on wide dental arcades. Functional examination failed to reveal any hearing loss.

The palpebral fissures showed a gentle antimongoloid obliquity. On the right side, the inner canthus was partially hidden by a cutaneous fold, as in blepharophimosis. The interpupillary distance amounted to 65 mm. The skin of the lids and the upper tarsi had a normal appearance; the lower tarsi, on the contrary, were markedly narrow, thin and pliable. The palpebral border presented a small indentation in its lacrimal segment, and the lower lacrimal papilla, punctum and canaliculus were absent. The upper papilla was well conformed, in spite of which no traces of the upper punctum were found. No lacrimal fistulas could be demonstrated. Tearing, which was permanent, was accounted for by the atresia of the normal drainage system.

A permanent right esotropia of 6△ and a hypertropia of 4△ were registered when the screen-prism test was performed at a distance of 20 feet. In direct elevation, the esotropia disappeared and was even replaced by a marked exotropia, while in direct deorsum-

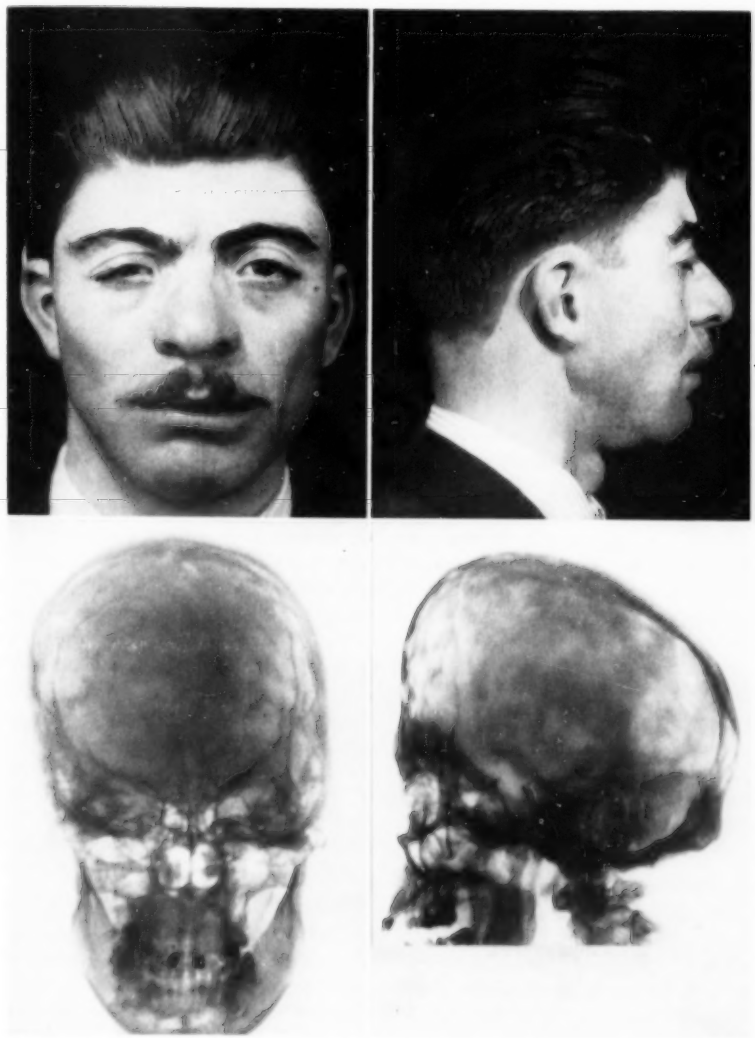

FIG. 6.—Isolated case of the same malformation occurring in association with a pronounced acrobrachycephaly.

version it became more accentuated: divergence in elevation and convergence in depression.[38] Worth's four-dot test revealed alternating suppression. A most conspicuous overaction of the right inferior oblique was present, as well as a slight degree of right palpebral ptosis. No exophthalmos existed.

The media were normal but the fundi exhibited, as in many cases of oxycephaly,[45] some medullated nerve fibers. Paramacular retinoscopic findings were as follows: O.D., + 0.50 cyl., ax. 90°; O.S. + 0.50 cyl., ax. 15°. Uncorrected vision amounted to 20/80 in the right eye and to 20/30 in the left one.

Roentgenologic findings were consistent with a dyscephaly ranging midway between oxycephaly and craniofacial dysostosis (fig. 6). The cranial vault was reduced in its transversal and sagittal diameters; the vertical diameter instead, was increased at the expense of a large bregmatic bosselation, which evidently resulted from a compensatory overgrowth of the anterior fontanelle. All suture lines were obliterated and well developed digital impressions were present. The sella was enlarged in all diameters. The orbital roof showed a rather sharp slope. Sphenoidal angle: 122 degrees (basilar kyphosis); nasal angle: 66 degrees; maxillary angle: 96 degrees.

Although congenital, the deformity was in this case neither hereditary nor familial; yet, it should undoubtedly be regarded as germinal and embryonic in origin.

CAUSAL AND FORMAL GENESIS

It is a well-known fact that the bony walls of the orbit result from condensations occurring in both the visceral and the paraxial mesoderm; the lower and outer walls are formed from the maxillary process, in which the malar bone and the maxilla (except its frontal process) are developed; the upper wall is formed from the capsule of the forebrain, in which the frontal bone develops, and the medial wall derives from the lateral frontonasal process, which differentiates into the frontal process of the maxilla and the lacrimal and nasal bones.

The upper and the lower lids develop originally as ectodermic folds from the frontonasal and the maxillary processes, respectively; the mesenchyma which at a later stage grows in to form the tarsal plates issues from the paraxial mesoderm in the case of the upper lid and from the visceral mesoderm in the case of the lower one. The lacrimal passages develop out of a solid rod of ectodermic cells which separate from the surface and are buried as the maxillary process, rapidly growing toward the midline, overlaps the lateral nasal process; the canaliculi and puncta are the result of outbuddings from the upper end of this rod, which finally canalizes in the 35 mm. embryo.

On the other hand, the sclera, the choroid and the funnel-shaped premuscle mass from which the extraocular muscles derive, originate from the paraxial mesoblastic layers encircling the optic cup.[3,8,23b]

Accordingly the multiple malformations which integrate the syndrome under consideration must be attributed to a failure of the mesoderm of the first, premandibular somite to differentiate properly into the corresponding osseous, connective, vascular and muscular structures. As the paraxial mesoderm is in close continuity and fuses with the visceral mesoderm of the maxillary process, which limits anteriorly the first branchial cleft, it is not surprising that any noxious influence affecting one of them may disturb the other.[39] In that way, not only the bony deformity but also the tarsal plate agenesia, and even the palpebral ptosis, the isolated underaction of the superior rectus and the hyperopia itself could be adequately accounted for. It is to be noted that in one case in particular, the mother in family B (fig. 4, II), the coincidental occurrence of a large amount of mixed astigmatism and of a slight palpebral ptosis pointed strongly to a faulty development of the orbital mesoderm.

In mandibulofacial dysostosis, in which the paraxial mesoderm is ordinarily spared, as are also the structures derived from the lateral frontonasal process, the malformation affects predominantly the external two thirds of the maxillary process and the dorsal surface of the first branchial cleft, thence resulting in the all too well known agenesia of the zygomatic bone, the lids and the external ear. In craniofacial dysostosis, the whole maxillary process and the medial nasal process are ordinarily involved, the consequence

thereof being the hypoplasia of the whole upper maxilla and of the intermaxillary bone and the abnormal brevity of the upper lip.[42]

The lacrimal abnormalities represent a disturbance in the fusion of the maxillary and the lateral nasal processes rather than an arrest in development of the processes themselves, in the same way that a cleft lip and a cleft palate are the result of an imperfect fusion of the maxillary and the medial nasal processes. Among these abnormalities were represented the three main types of developmental defects affecting the passages described by Duke-Elder[9]: (1) Changes due to an abnormal course being followed by the budding cells which form the canaliculi; to these must be added the appearance of a true third canaliculus as the result of a supernumerary budding-out of the upper part of the primitive epithelial column. (2) Failures in canalization, resulting in atresia. (3) Changes due to failure in the regular fusing of the lateral nasal process and the maxillary process. The latter were present in the second family and in the isolated case just mentioned as a minute coloboma of the innermost part of the lower lid margin, which should be looked on as an abortive form of facial cleft.

On the whole, the syndrome consisting of agenesia of the orbital margin, hypoplasia of the tarsal plates and of the skin of the lids, developmental errors of the lacrimal passages, medium grade hyperopia or astigmatism and, eventually, even of defects of the extrinsic ocular musculature, must be regarded as a different and this time indubitable expression of that developmental correlation which was supposed to exist between certain parts of the eye and its neighboring structures, on one hand, and the facial skeleton on the other,[24] and which, in the case of the size of the cornea and of that of the mandible, has been questioned recently by Hallermann.[19]

As shown by the pedigrees of the affected families and even by the type of the aberrations themselves, all doubts concerning the germinal nature of this multiple malformation must be discarded at once. Yet, two different possibilities should be taken into account as soon as the chromosomal mechanism involved in its hereditary transmission is subject to closer consideration. Of these, one would ascribe the diverse defects constituting this syndrome to the action of several genes located in the same chromosome. The other, by far the most plausible,[13] would conclude from the occurrence of these manifold, related abnormalities, that one is dealing with a unique gene of a *pleiotropic, polyphenic* nature, which influences the embryo at the 8 to 16 mm. stage. This gene is similar in its mode of action to those held responsible for Crouzon's cranio-facial dysostosis, for mandibulofacial dysostosis, and for the deep skeletal and muscular deformities that are frequently found in association with developmental defects of the external ear.[17]

The fact that one patient of the first family (fig. 1, III, 3) exhibited only an incomplete form of the affection ought to be explained on the assumption that the mutated gene *expression* may display eventually some slight variations, for the possibility that crossing-over would have broken the linkage existing between two or more genes occupying the same chromosome seems extremely remote.

On the other hand, the scarce qualitative variability shown by the syndrome points to a high specificity of the character concerned.[21] Both the dominant mode of transmission and the elevated penetrance rate of the anomaly are clearly demonstrated by the pedigrees presented.

The fact that no case showing the syndrome died immediately after birth or in infancy, as well as the absence of any history of miscarriages, reveals that the mutated gene has no lethal effect.

DISCUSSION: RELATIONS TO OTHER DEVELOPMENTAL DISORDERS, PARTICULARLY TO OTHER FORMS OF CEPHALIC MALFORMATION

Agenesia of the orbital margin and even of the orbit as a whole[23a] is of common occurrence in cases of oxycephaly, of plagiocephaly, of acrobrachycephaly (to which the cephalic deformity found in acrocraniosyndactyly corresponds) and, indeed, of most dyscranias, where, in addition, intracranial hypertension depresses the orbital roof.[29] The same holds true in cases of Crouzon's craniofacial dysostosis.

In mandibulofacial dysostosis, a certain hypoplasia of the orbital margin, and of its zygomatic section in particular, is also encountered, as it is in cases of strabismus in which a congenital insufficiency of one or both superior rectus muscles is found.[39]

Conversely, in cases of Villaret and Desoille's familial primitive hypoplasia of the upper jaw,[40] where an extreme underdevelopment of the malar and maxillary bones is said to exist, the orbital region, or rather that part of it which is formed by the frontal bone, would be notably prominent.[27]

Hence, we see that a total or segmentary aplasia of the orbital margin must be expected to occur whenever the corresponding embryonic mesodermal structures are involved in a widespread dysontogenic process; the ensuing developmental error would then be either secondary to the action of mechanical forces resulting from distant deformities, or primary and due to an intrinsic inability of the mesoblastic anlage itself to differentiate properly. No attention seems to have been given, however, to the possible occurrence of an isolated or almost isolated hypoplasia of the orbital rim which, as in the cases presented, should be regarded as arising from a localized arrest in development.

The hereditary character of congenital defects of the lacrimal passages has been early recognized, and true instances of a dominant mode of transmission are many.[32] It is interesting to note that, though rarely and as an accidental finding, such anomalies have been encountered in some cases of diverse cephalic malformations. Thus, in O'Connor and Conway's two cases of typical mandibulofacial dysostosis,[26] the lacrimal puncta were not apparent. In Waardenburg's incomplete case of the same syndrome,[44] an absence of the lower puncta was recorded.[14] In another incomplete form observed by Roussel,[30] the whole lower canaliculus was absent. On the other hand, an abortive facial cleft in the form of a coloboma of the right nostril was present in Tranos' second case of atypical Franceschetti's syndrome.[36] Absent puncta were also noticed by Anderson[1] in two cases of acrocraniosyndactyly. Hoffmann-Egg and Velissaropoulos' third case of associated ocular and auricular malformations[20] presented a minute coloboma of the innermost part of the right lower lid, as well as a chronic dacryocystitis on the opposite side; these authors' first case exhibited, likewise, a purulent dacryocystitis on the left side; in neither of them, however, were the lacrimal passages adequately explored.

Anderson and Woodhall's first case of Crouzon's malady[2] (fig. 7) had in common with our patient M. P. (fig. 2), whom she resembled vaguely, the abnormal flatness of the orbital margin and the reduced values of the sphenoidal, nasal and maxillary angles (122

FIG. 7—Familial craniofacial dysostosis of Crouzon (reproduced, with permission, from Anderson and Woodhall[3]).

degrees, 48 degrees and 72 degrees, respectively). Nevertheless, their case differed widely from ours in that it exhibited the whole typical picture of craniofacial dysostosis, without showing any palpebral or lacrimal anomaly.[1] In Vogt's second case of dyscephalodactyly,[42] which was published again by Nager and deReynier[25] as their case no. 4, a conspicuous aplasia of the orbital margin existed, which by vitiating exophthalmometric readings invalidates partially the conclusion that a high-grade proptosis was present; although no special data are given on the status of the tarsal plates and of the lacrimal passages, these can be supposed to have been normal, for any such aberration would not have escaped the attention of the Swiss investigators.

FIG. 8—Dyscephalodactyly of Vogt (reproduced, with permission, from Nager and deReynier[25]).

According to Sorsby,[34] a dark coloration of the skin of the lids with the dermic veins standing out markedly has been observed as a dominant affection over five generations. Yet, I have found no mention in literature of the occurrence of a cutaneous hypoplasia circumscribed to the lids. Aplasia of the tarsal plates has been in turn observed by Aubineau[4] as a transmissible isolated deformity in a mother and her two children, all three of whom suffered from entropion.* Similarly, among the many palpebral abnormalities recorded

*The relationship existing between congenital anomalies of the extraocular muscles and some definite, slight facial malformations has been thoroughly reported by me in a recent publication.[39]

in Waardenburg's syndrome of embryonic fixation,[43] underdevelopment of the tarsi has been noted by Marchesani.[24]

As demonstrated by the preceding review of the pertinent literature, each of the main constituents of the syndrome under consideration has been reported to occur sporadically or as an inherited character, either alone or in conjunction with different cranial or facial deformities. Nothing needs to be said in addition on the well known hereditary transmission of hyperopia, palpebral ptosis and isolated congenital palsies of the extraocular muscles.

To the best of my knowledge, however, the association of the above mentioned defects in the form of a well defined, hereditary clinical entity has hitherto not been described, be it as a strictly isolated developmental error or in combination with minor or major cranial abnormalities.

CONCLUSIONS

Although the condition was named after its main characteristic as *familial hypoplasia of the orbital margin*, no claim is made as to its being any essentially new or different type of craniofacial dysostosis.

I contemplate it merely as a germinally determined complex of symptoms that, though casually associated, have a common pathogenetic basis.

This symptom complex, which responds to a clear-cut hereditary pattern, may occasionally be seen to merge into other more or less widespread forms of cephalic aberration. Such indeed is the tendency with all forms of facial and mandibular dysostosis and with Crouzon's disease, whose boundaries are sometimes hard to establish.

SUMMARY

A rare, hitherto undescribed developmental craniofacial disorder of a high familial incidence is described, the main features of which are (1) a marked agenesia of the orbital margin, (2) a polymorphic lacrimal anomaly and (3) an aplasia of the tarsal plates and of the skin of the lids. A variable amount of hyperopia is frequently present, as well as some congenital defects of the extraocular muscles.

A faulty development of the paraxial and visceral mesoderm which differentiate into the osseous scaffolding of the upper part of the face is considered responsible for this malformation; the accompanying errors of the overlying and surrounding soft parts could be brought about by the same noxious process, which afflicts the embryo at an early stage. The hyperopia encountered may result from a failure of the mesenchyma which encircles the optic cup and derives from the paraxial mesoderm to respond properly to the stimulus represented by the growth of the retina. The insufficiency of some of the extraocular muscles also depends on a defective differentiation of that part of the paraxial mesoderm from which the premuscle mass comes forth.

The condition is transmissible as a dominant, most penetrating character, as shown by the pedigrees of two identically affected families. Only minor quantitative and qualitative variations in the effect of the mutated gene were recorded (fairly constant *expression* and *specificity*).

Diagnostic considerations are made, regarding slight forms of Crouzon's craniofacial dysostosis, Franceschetti's mandibulofacial dysostosis, Villaret and Desoille's familial hypoplasia of the upper maxilla and Franceschetti's cranial dysostosis with cerebriform calotte.

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DISCUSSION

HAROLD F. FALLS, M.D., Ann Arbor, Mich.: Dr. A. Urrets-Zavalia's excellent presentation of an apparently unrecognized syndrome is deserving of high commendation. To recapitulate, this interesting syndrome includes the following features: (1) underdevelopment of the orbital margins, (2) anomalies of the nasolacrimal ducts, (3) maldevelopment of the tarsal plates of the eyelids, and (4) hypoplasia of the skin of the eyelids. An increased incidence of hypermetropia, as well as associated strabismus, also suggests a possible relationship of these features to this syndrome.

The etiology of Dr. Urrets-Zavalia's complex of developmental facial anomalies is apparently genetic, being determined by a pleiotropic autosomal dominant gene. The gene presents, in these two families, excellent penetrance but does exhibit, not withstanding, a considerable variability of expressivity. Primary hypoplasia of the orbital margin, like that of its closely allied syndrome, namely, mandibulofacial dysostosis, is the result of the deleterious effect of a noxious gene acting (and let me emphasize this) at a specific time interval in the development of the paraxial and visceral (the latter being maxillary) mesoderm. The subsequent developmental aberration of this mesoderm results in disturbances of the adjacent adnexa and in this syndrome produces anomalies of form or function of the skin of the eyelids and of the lacrimal passageway. In this respect I should like to inquire of Dr. Urrets-Zavalia if he observed any anomaly of form or function of the orbicularis oculi other than for abnormal insertion of the canthal ligaments.

Classification of the developmental anomalies of the skull and face defy orderly presentation. As mentioned, yet not accepted, by the author, Sir Stewart Duke-Elder suggests that much of the developmental abnormality of the skull and face "appears merely to be a result of a variable anatomical localization of the same fundamental disturbance." I should like to modify Sir Stewart's concept and suggest that the fundamental disturbance is gene determined and that the phenotype that results is the complex end result of a considerable number of features. These include (1) specific time and length of time that the noxious gene action occurs during the embryological development of the key anatomical structure, (2) maternal environmental agent action (varies for each individual), (3) genetic constitution of each individual (always unequal except for identical twins), and (4) individual anatomical variation (also gene determined).

Individual variability in expressivity of the syndrome features could thus result from variation in any one or all of the above-mentioned factors. It is also easily conceivable that intermediate forms between different syndromes could result. It must be emphasized that noxious maternal environmental agents, such as rubella or toxoplasmosis, could produce identical similar syndromes (phenocopies) providing the deleterious action occurred at the same specific time interval in embryological development.

I should like to thank the Program Committee for the privilege of discussing Dr. Urrets-Zavalia's paper and to praise the author for bringing to our attention this most interesting anomalous facial syndrome.

DR. URRETS-ZAVALIA: In the first place I must thank Dr. Falls for his kind and most conservative discussion, but I should say I could not have expected less from my American colleagues and from him.

As to his questions, I will only say that in mandibulofacial dysostosis the paraxial mesoderm is ordinarily spared, as are also the structures derived from the lateral frontonasal process. The malformation affects predominantly the external two thirds of the maxillary process and the dorsal surface of the first branchial cleft, thence resulting in the all too well known agenesia of the zygomatic bone, the lids and the external ear.

Of course, the possibility of transit forms between the syndrome I described and Franceschetti's syndrome may exist, and in closing my paper, as the last conclusion, I said:

"This symptom complex, which responds to a clear-cut hereditary pattern, may occasionally be seen to merge into other more or less widespread forms of cephalic [illegible]stion. Such indeed is the tendency with all forms of facial and mandibular dysostosis and with Crouzon's disease, whose boundaries are some times hard to establish."

As to Dr. Falls' question concerning the paper I presented at the International Congress of Ophthalmology, I would say that I was not referring then to facial asymmetry, which is a very well known fact and which is always taken into account by anyone concerned with the study of craniofacial defects, and that neither asymmetry nor major orbital malformations were seen. They were only minor, almost subclinical, malformations which were referred to at the time.

No anomaly of form or function of the orbicularis oculi was detected in my cases, the abnormal prominence of the inner canthal ligaments being secondary to the underdevelopment of the underlying osseous structures.

And to conclude this discussion, I must say that I am in complete agreement with Dr. Falls as to the possibility for any noxious influence acting upon the embryo at the 8 to 16 millimeter stage to bring about this malformation. However, I have never seen nor heard about such a condition occurring as a consequence of a peristatic factor; in these cases at least it was definitely genotypical in nature, since it was transmitted over three generations.

Thank you very much.

A CURRICULUM OF OTOLARYNGOLOGICAL UNDERGRADUATE AND POSTGRADUATE INSTRUCTION IN PROSPECT

A. C. Furstenberg, M.D.
ANN ARBOR, MICHIGAN

The demanding disciplines of medicine today call for a broad general well-integrated knowledge of many fundamentals of the basic sciences and clinical medicine. Any attempt to list them here would be superfluous. As a whole, they constitute the sum total of that knowledge essential to the practice of medicine. New discoveries in the various fields of science cause rapid changes in our educational programs but the basic subject matter of a modern curriculum is governed in a very large measure by public demand.

I should like to say with all the seriousness of which I am capable that otolaryngology is one of the fundamentals of a broad general knowledge of medicine. No doctor can conduct the general practice of medicine with safety to his patients without a practical knowledge of the common diseases of the ears, nose and throat. My experiences of many days of practice during vacation periods in the offices of general practitioners in metropolitan areas and in rural communities convince me that otolaryngological diseases constitute a very significant part of the general practitioner's daily work.

It is discouraging to observe what has happened to otolaryngology in the curriculums of some of the leading medical schools of this country in the past decade. New subject matter in the basic sciences and a growing appreciation of psychological, sociological, and environmental factors of medicine have pushed otolaryngology out into the periphery of the curriculum in a number of medical schools, where it is dangling either in obsolescence or without the support of integration with the other important components of an educational program. But there are some encouraging trends in a few of our medical schools today that lead me to be optimistic. I believe we are emerging from a period of decadence and that renewed interest in and support of otolaryngology in institutions where it has been neglected is in the offing. Investigators in this field have been prolific in their discoveries and are rapidly bringing to otolaryngology scientific facts which must be incorporated into a modern medical curriculum and put into practical usage. These findings are being evaluated by some educators with enthusiasm and an appreciation of their importance. Moreover, the American public is demanding more and better service in this field.

What we need now is to recruit new leadership in academic otolaryngology. We must have teachers who realize that their responsibilities reach beyond those of solving mere technical problems and that they must inspire the student and emphasize to him the possibilities of otolaryngology. The men of prestige and leadership of national scope in our field are the strong, resourceful individuals who have developed comprehensive and effective programs of instruction which command student attention and respect. The proper time to

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impress the student and to stimulate his interest in our field is during his early formative undergraduate years in the medical school.

Come now to what might be considered a minimum undergraduate curriculum of otolaryngology. I realize that it must necessarily vary in different medical schools in order that it may function as an integrated part of a well-integrated educational program. In this connection, I hasten to say that no individual or agency can prescribe a schedule of study which is ideal or even moderately successful for all institutions. The effectiveness of any program of instruction will invariably depend upon the competence and interest of its leadership.

Before attempting to outline a course of undergraduate otolaryngologic instruction, I want to dwell briefly upon a fundamental pedagogical principle which I fear is often neglected but is nevertheless exceedingly important to any acceptable program of instruction. It pertains equally importantly to our programs of residency instruction.

The time-honored and well-established system of medical education thrusts the student into two years of arduous study of the basic sciences and then abruptly transfers him to a study of the practical clinical problems in the hospital. In short, there is a wide gap between the basic sciences and clinical medicine. The former are divorced from the latter with the result that our upper classmen are often woefully deficient in their knowledge of the fundamental sciences. The fault lies not in the time and energy given the fundamental sciences because our students know them in all of their minutia immediately upon completion of the courses and after their review for the final examinations. The trouble rests in a failure to correlate the basic sciences and clinical medicine, in short, in the omission of an effort to give the basic sciences a practical application by integrating them intimately with clinical medicine.

At the University of Michigan, we commence our undergraduate studies in otolaryngology in the junior year. At this time the student is returned to the anatomical laboratory, where he studies the anatomy of the ears, nose, and throat by dissection, lectures and quizzes. Concurrently, he is introduced to some genuine practical clinical problems in the hospital. Thus we observe that student interest and enthusiasm run high when anatomy is taught to enlighten and elucidate clinical otolaryngology.

Ten hours of anatomical study of this type are given each member of the junior class. They do not add time to the medical curriculum because these fields are not studied during the regular course in anatomy in the freshman year. They are purposely avoided the first year in order that they may be intimately correlated with clinical otolaryngology at a later period of training without expanding a medical curriculum which is already overcrowded and cumbersome. We see hopeful signs of the student's ability to retain a working knowledge of anatomy of the ears, nose, and throat at the time of his graduation which capacity we did not observe in earlier years when he was divorced from the subject at the termination of his freshman studies.

The junior medical students also receive 32 hours of demonstrations and lectures which teach the technique of examination and cover the entire field of otolaryngology as it pertains to the general practice of medicine. Approximately one-half of this time is spent in becoming familiar with examining meth-

ods and learning how to see the ears, nose and throat. Carefully compiled lecture notes covering the principal diseases in this field are provided in order to cover the subject thoroughly and to conserve teaching time. Instruction is given in the functional examination of the ears, and two lectures are offered in the physiology of acoustics. These formally scheduled exercises are held at the time in the curriculum when the student is also exposed to courses in pathology, pharmacology and radiology, and immediately after he has completed a most comprehensive study of the physiology of respiration.

Thus, the student comes to us in the senior year with the ability to see the anatomical regions in which we are interested and with a fairly comprehensive understanding of the common otolaryngological conditions gained from 32 lectures and a thorough study of the mimeographed data which are placed at his disposal. He has already successfully passed his junior examinations on the technique of observation and the content of the lectures and notes.

All of our senior medical students experience a minimum of 80 clock hours of instruction in the form of informal seminars, out-patient examinations and ward rounds. The class is divided into sections of eight to ten students who spend ten full days in the Department of Otolaryngology. At the first period each morning an informal conference, attended by all students in the section and the resident staff, is held by one of the professors in the Department. The conference continues for one hour and a half. Cases which have been assigned to the students the day before and worked up by them during extracurricular hours are presented for discussion as to history, physical findings and treatment. Prevention is stressed in every instance when relevant. The members of the resident staff as well as the senior students enter freely into the discussions in order to bring out all pertinent data and to give widest possible consideration to all phases of diagnosis and therapeutic management. In this manner an effort is made to make the conferences interesting and stimulating to both undergraduate students and residents.

Immediately after the conference, the students are assigned to the out-patient clinic or to ward work for the remainder of the day. In the dispensary, they examine patients and confer with senior members of the resident staff in regard to diagnosis and treatment. On the wards, the students are required to make a complete physical examination of all new patients and are supervised and instructed at the bedside by the senior members of the staff. No didactic lectures are presented in the senior year and no time is spent in the operating rooms.

It is gratifying to observe that after a period of concentrated study for ten days, our students show considerable aptitude in the technique of examination of the patient and they carry away with them an appreciation and understanding of the types of patients which they are likely to see in a general medical practice. Every effort is made not to squander the student's time with a consideration of rare otolaryngologic problems or with the demonstration of major surgical techniques.

It is possible to capture the interest, enthusiasm and imagination of the undergraduate medical student in otolaryngology if he is given contact with patients in the dispensary or wards and invited to participate in a study of their problems and care. Academic leadership of a stimulating type is, of course, most essential in gaining these ends.

RESIDENT INSTRUCTION

In any discussion of continuing education, one must refer to that program which is designated as graduate study and calls for a prolonged period of instruction in an educational institution after the internship has been served. It implies concentration in some particular field—research, perhaps, into the reasons for certain methods of practice—and frequently leads to the Master's degree for those who have satisfactorily completed prescribed requirements. It is the highly accepted and approved method of acquiring the knowledge and technical skills of the specialties and the program for which the majority of graduates of medicine invariably strive. Fortunately, this opportunity is open now to nearly all who desire specialized training in otolaryngology and obviously can be offered in its consummate fulfillment in many teaching institutions in this country.

To be specific, this curriculum of continuing education in otolaryngology calls for a rotation service which creates a staff ranging from the assistant resident at the bottom of the class to the oldest individual in years of training who is approaching the termination of a three or four year term of study. In this group there are staff members with varying degrees of experience and skill who transmit their knowledge of the subject to those whom they outrank and receive instruction in turn from the superior officers on the staff. Thus, each member of the group assumes the function of an instructor and the responsibilities thus imposed upon him are invariably followed by a diligent search for knowledge and information which he may pass on to those whom he is expected to serve.

The maintenance of a rotation service affords an opportunity for staff members to work shoulder to shoulder each day, to give their knowledge, experience and technical instruction to their subordinates and to receive the compensatory benefits of superior training and judgment from their elder colleagues. Groups of young men on rotation services of this type, however, must be inspired, diligently instructed and supervised by senior physicians in the department who have had years of experience in teaching and the practice of otolaryngology.

The residency in Otolaryngology at the University of Michigan is of four years duration. After completing an approved internship, the appointee for the first year is given the rank of Assistant Resident. During the first three months of his service in this capacity, he deals with in-patients in the hospital in order that he may become familiar with preoperative and postoperative care. He is then transferred to the dispensary where he is instructed daily by senior residents in the diagnosis and therapeutic management of the patients who visit the out-patient clinic. Early in his career he is given an opportunity to perform some of the minor surgical procedures in this field.

In the second year of his service, he is given the rank of Resident and assumes greater responsibilities in the out-patient clinic and operating rooms. The third year he is appointed a Junior Clinical Instructor and assigned teaching duties in anatomy of the head and neck for junior medical students. Finally, in the fourth year, he obtains the rank of Senior Clinical Instructor and is held responsible for one fourth of the instruction given senior medical students and graduate students.

During the four years of continuing education in otolaryngology, the basic sciences are taught the resident in intimate correlation with clinical otolaryngology. Formal courses are offered in

pathology, anatomy of the head and neck, and radiology, but only in connection with clinical work. Each day pathologic sections of tissue obtained from regions of our particular interest are sent to the Department of Otolaryngology for review and study by members of the staff. Embryology is taught in an applied manner as it relates particularly to the many teratological tumors and cysts which are observed in the head and neck.

The service deals in a comprehensive manner with principles of surgery. During the four-year period, the resident obtains a thorough training in surgery of the ears, nose and throat, bronchoscopy and esophagoscopy, and many of the radical procedures in the neck essential to the treatment of malignant disease and congenital anomalies. Special attention is given to diseases of the salivary glands and instruction in rhinoplasty and otoplasty is available to those who are interested in these procedures.

We all agree, I am sure, that where there is no research there can be no progress in medicine. The members of our staff are well aware that the complexities of medical research call for cooperative effort today and that, as individuals, by personality and temperament they must be well equipped to work as a team. Today, of necessity, research is a collective enterprise in which not one individual but many enthusiastically unite. We request each member of the staff to work on one of these teams. Thus, we find residents in otolaryngology collaborating in research studies with the neuro-anatomists, radiologists, endocrinologists, pathologists and a number of investigators in fields remote from otolaryngology. In this connection, I should like to mention that one of our residents is collaborating with a geologist in the study of salivary calculi with resulting discoveries which I think will be of considerable interest to some members of this audience when they are published.

And finally, while instructing our undergraduate students and residents, let us not overlook their training and preparation in the sociological, psychological and environmental aspects of medicine. It is vitally important that our students learn these essentials of practice: a knowledge of human conduct and behavior, an understanding of mental and physical reactions to the emotional influences of life, and appreciation of the variable human capacities for enduring pain and sorrow, and resisting adversity and despair. All these factors comprise the very essence of medical practice and the success of the doctor today is dependent in a very large measure upon his recognition and understanding of these exceedingly important causes of illness.

The young doctor must realize that much of the future practice which he hopefully anticipates will consist of patients who are experiencing some social, economic, moral or religious conflict, that their physical ills are a reflection of some emotional state and that drugs and operations will not cure them. From the day premedical education commences to the hour of graduation and even beyond that point in the field of postgraduate instruction, the training of the doctor is predominantly in the organic sciences. A pain in the head is the result of sinus disease, brain tumor or cerebral arteriosclerosis and if the student cannot find an organic basis for the headache, he loses interest and passes on to his next assignment. Seldom is it impressed upon him, or if the effort has been made the impression did not "take," that the headache in all probability is due to some emotional conflict just as important as an organic disease; and, when therapeutic measures

of a proper psychologic character are instituted, the patient has a good chance to recover.

In closing I should like to emphasize that there is a great demand for otolaryngologists in this country today. The field is exceedingly barren. If anyone doubts this statement, I call attention to an experience one of my senior staff members had last year. This young man, about to complete his training, advertised with an ethical professional employment agency and received more than two hundred invitations to practice otolaryngology in widely distributed geographic areas over the entire United States. Invariably, however, Board certification is required. We must exert every influence to establish comprehensive programs of otolaryngological instruction in our undergraduate curriculums in order that we may properly indoctrinate the undergraduate student and provide the incentive for continuing education in this field. May we likewise proceed resolutely to mobilize all of our resources in the interest of adequate facilities for postgraduate instruction in this essential surgical specialty. Let us not fail in our solemn obligation and responsibility to educate well-qualified otolaryngologists to render the type of service urgently required today by all groups of our population, economic as well as geographic.

EMBRYOLOGY OF THE EAR

A motion picture in color with sound

Sponsored by

American Academy of Ophthalmology and Otolaryngology

Dr. Franz Altmann and the Audio-Visual Committee

Produced by Sturgis-Grant Productions, Inc.

16mm • 1332 feet • Showing time: 1 hour, 32 min.

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100 First Avenue Building

ROCHESTER, MINN.

THE USE OF SKIN GRAFTS ABOUT THE HEAD AND NECK

WILLIAM C. HUFFMAN, M.D.
DEAN M. LIERLE, M.D.
IOWA CITY, IOWA

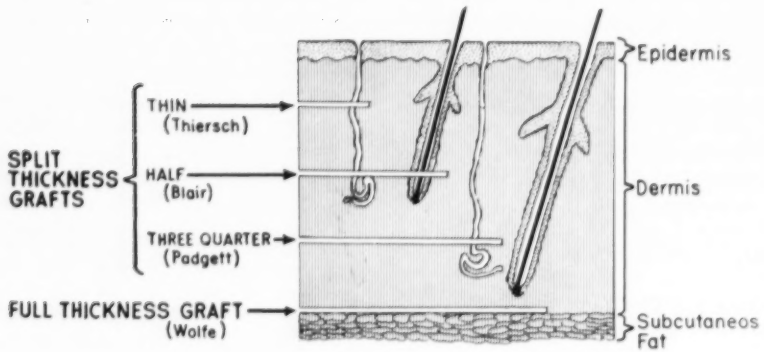


FIGURE 1.

THE principles covering skin grafting are the same regardless of the part of the body in which the grafts are used. There seems to be a rather widespread belief that something mysterious or hazardous accompanies the use of grafts. As a result, complicated and time consuming flaps are often employed in order to avoid the possibility of losing a graft.

Some of the fundamental features concerning grafts can be grouped under the following headings:

1. Nutrition
2. Expected purpose
3. Thickness
4. Size
5. Application
6. Fixation
7. Tension

State University of Iowa Hospitals, Iowa City, Iowa. Presented at the Fifty-Ninth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Sept. 19-24, 1954, New York, N. Y.

The nutrition of a graft proceeds through several stages. For the first few days after its application the graft depends upon plasma from the recipient bed. A little later there are anastomoses between the vessels of the bed and those of the graft along with ingrowth of new vessels into the graft. It is well to remember that with the possible exception of the periphery of the graft these vessels grow into it in a perpendicular manner. This arrangement is permanent. Later attempts to elevate or transfer the graft as a flap without provision of enough deep tissue to furnish sufficient blood supply to it will result in its death.

Except in extremely rare instances a skin graft serves only one purpose, the provision of a covering or a lining. An analogy might be drawn between a graft and ordinary wallpaper. The paper merely serves as a cover and has little or nothing to do with the form or stability of the wall. Although ultimate deep tis-

FIG. 2—Infected granulation tissue demanding earliest possible cover by use of thin skin grafts.

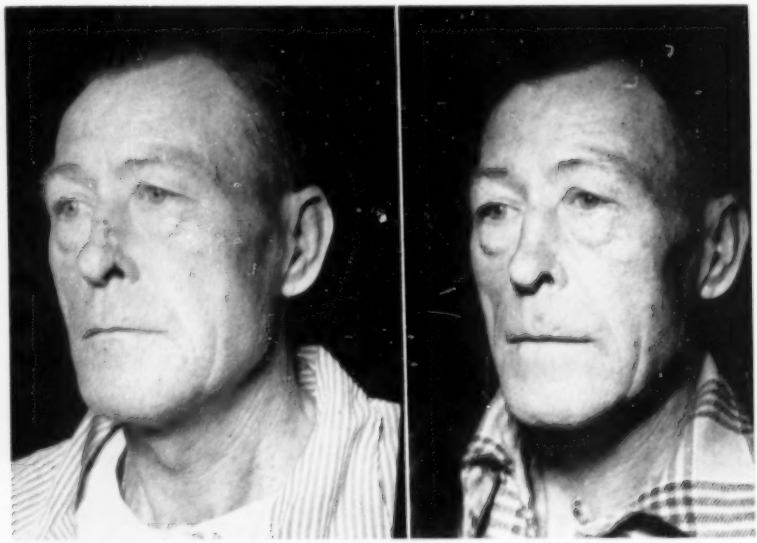
FIG. 3—Postirradiation ulcer. Following excision there is usually a defect with blood supply that is insufficient to support any graft other than a very thin one.

sue changes may result in late contour improvement, it is not judicious to rely solely upon a graft when a defect that has an appreciable subcutaneous component is to be corrected.

Extremely thin grafts (fig. 1) "take" well but are the ones most apt to undergo undesirable contracture and color change. The reverse is true when thick grafts are used. Very thin grafts find their usefulness in instances where a "take" is imperative or where it is unwise to have the deeper tissues covered by a heavy pad of tissue. For examples, infected granulations (fig. 2) and regions of poor capillary supply (fig. 3) usually will not support a thick graft; thin grafts are indicated after excision of a malignancy (fig. 4) in order to allow early detection of recurrent or residual neoplasm.

Thick grafts, either thick split thickness or full thickness, are in order when the cosmetic result is of prime importance. Corrections of contractures and skin replacements after excisions of benign skin lesions call for thick graft re-

FIGURE 4.



A B

FIG. 5—(A) Nasal scar. History of recurring ulceration upon exposure to extreme cold; (B) Result of scar excision and repair with thick skin graft.

pair in the hopes of preventing shrinkage and of obtaining good color and texture (fig. 5).

One well established principle is that all skin grafts shrink. Thick or thin grafts undergo shrinkage for about a month after application. After this length of time the thin grafts are apt to shrink even further (fig. 6) while thicker ones expand to nearly their original size. Even when thick grafts are used it is sometimes disappointing to depend too much upon their regaining normal size. There is an often quoted statement to the effect that contractures of the lip, eyelid, or axilla are never corrected by one grafting operation. Careful consideration of the problems involved in such repairs leads one to the belief that probable graft contraction is not taken into consideration seriously enough at the time of application. If one overcorrects a soft tissue contracture by use of a much larger graft then at first appears necessary, he is not so likely to be plagued by partial recurrence of an ectropion or scar bridle (fig. 7).

FIG. 6—Degree of deformity that may result from shrinkage of a thin graft.

A B

FIG. 7—(A) Ectropion of lower lip caused by contracted scar; (B) Correction by scar excision and application of a large, thick skin graft.

As was stated previously, the life of a graft depends upon its ultimate vascularization from the recipient bed. It follows that a "take" depends upon intimate contact between every square millimeter of the graft and every square millimeter of the area of implantation. The necessity for such contact immediately demands a bloodless field and precise placing of the graft into every tiny crevice of the site to which it is applied. The belief that free blood beneath the graft helps in its nourishment is erroneous. Extravasated blood soon clots and then is only an obstacle to the growth of vessels into the graft.

The tension under which a graft may be sutured in place depends upon numerous factors, particularly the thickness of the graft and the character of the recipient area. A concave bed with an irregular surface may call for the graft to be laid in very loosely so that it may be in intimate contact with all parts. A convex bed with a smooth surface may allow the graft to be applied with considerable tautness. Very thin grafts seem to do about as well whether they are applied loosely or under noticeable tension. The tension upon thick grafts should approximate that of normal skin. It has been postulated that thin grafts do well under nearly any degree of stretch because the superficial vessels of the skin are without muscular walls and run parallel to the skin surface (fig. 8). Vessels deeper in the dermis are perpendicular to the surface and have muscular walls (fig. 8). Extreme tension reduces these deep vessels to slit-like compressed tubes, while extreme laxness allows their muscular walls to contract and greatly reduce the size of the lumina.

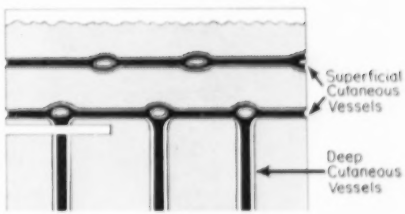


FIGURE 8.

After the previously mentioned intimate contact between graft and bed is attained, it must be maintained until the graft has had time to become vascularized, a situation comparable to holding pressure on a postage stamp until the glue has had time to become fixed. In the case of a graft this pressure must be kept for about five days and can be accomplished by the use of a tie-over dressing (fig. 9), careful bandaging,

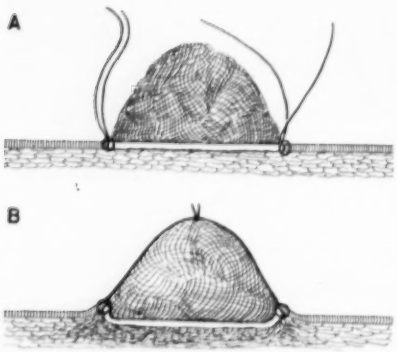


FIGURE 9.

or both. The degree of pressure applied should be about the same as that to which skin is subjected in its normal habitat. Application of proper pressure on the graft by tying sutures over a bolus dressing or by bandaging can be gained only with experience. Roughly it might be said that the correct degree of pressure is being exerted by tie-over sutures when the skin around the bulky dressing just begins to "tent." The proper pressure is being exerted by a bandage just when the sensation of "pulling back" of the bandage becomes apparent. One exception is the degree of pressure applied on a graft when it has been impossible to control oozing from the bed. Here the dressing must be tight enough to be hemostatic. Along this line a word of warning might be sounded

FIG. 10—Necrosis of most of graft, underlying tissue, and surrounding soft tissue as a result of excessive pressure exerted by dressing.

against the application of too firm pressure on a graft over a bony area. A little too much pressure collapses vessels against the unyielding bone and leads to necrosis of the graft and possibly of part of the bed (fig. 10).

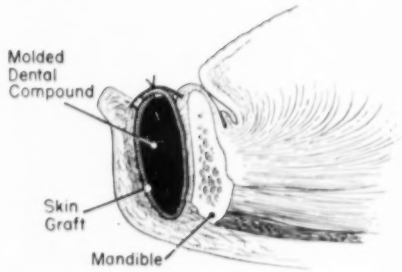


FIGURE 11.

The rules that are followed when skin grafts are used for surface coverings are tenable when grafts are employed for the lining of a cavity. Fixation is usually more difficult but can

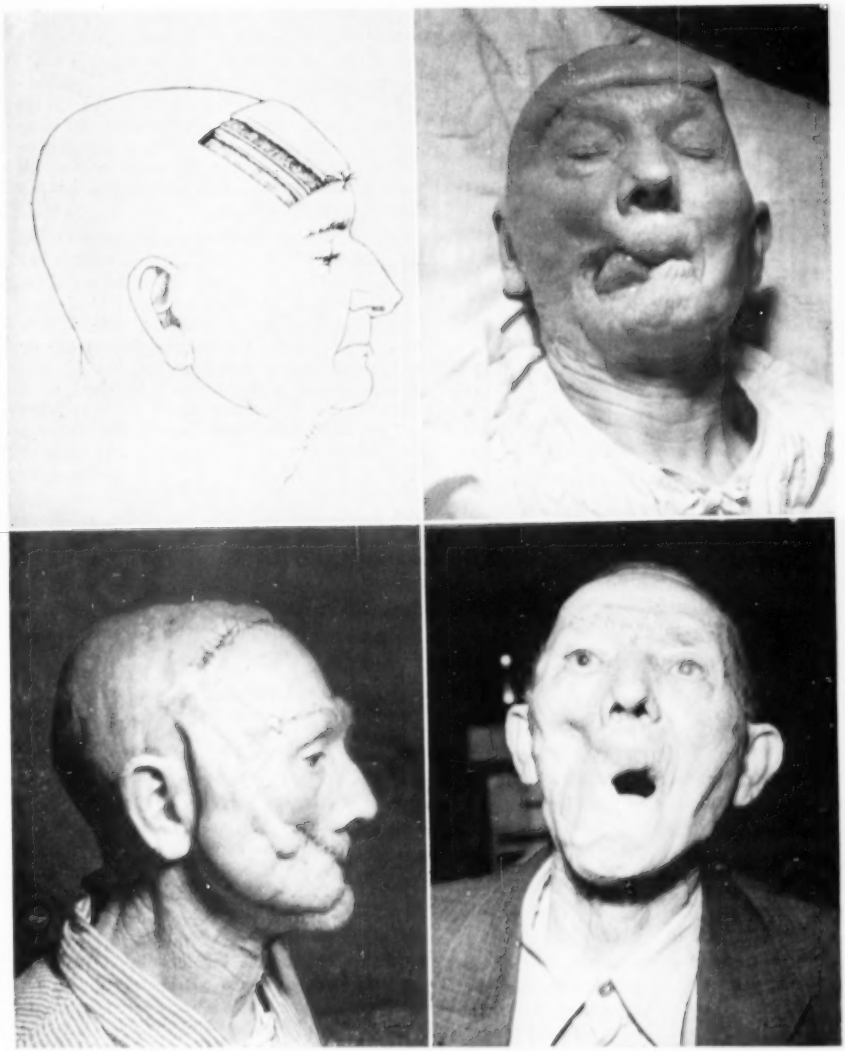


FIGURE 12.

ordinarily be obtained by use of molded dental compound (fig. 11) or foam rubber. Grafts put into the mouth should be taken from some relatively hairless area such as the inner surface of the arm. It is somewhat embarrassing to have a patient complain of long hair growing from the surface of his buccal cavity.

Through and through construction of a cheek or lip calls for a skin covering, a skin lining, and a generous amount of

soft tissue between the two. The covering and subcutaneous tissue can be gained by use of a flap from some satisfactory donor area such as the forehead. As a general rule it is best to apply the graft that is to provide the lining before the flap is moved to the defect. The use of two grafts face-to-face allows the flap donor area to be covered at the same operation (fig. 12). It is not good practice to bury only one graft to serve as covering for the donor area or lining for the flap. Unless grafts are buried with epidermal surfaces facing each other wound infection is apt to occur.

SUMMARY

The use of skin grafts depends upon a few basic mechanical and physiologic principles. As long as they are carefully followed successful results may be expected. Lack of attention to one or more of these fundamental rules usually proves to be the "weak link" that leads to loss of all or part of the graft.

DISCUSSION

EDGAR HOLMES, M.D., Boston, Mass.: It is always a pleasure to listen to Dr. Huffman's papers. Those that I have heard have been concise, to the point and complete. This one follows that pattern and leaves little to add or criticize. There are a few points that bear repetition and emphasis, just to drive them home.

As he stated in his final summary, in attempting a graft of any size, one must understand fully the physiology of wound repair and of graft reception and adhere to all the necessary criteria which have been mentioned in this paper. Doing this, one should obtain a satisfactory result.

Grafts as used by the rhinolaryngologist are mainly limited to the split graft as a cover for denuded areas left by the excision of malignant growths. Most other grafts, flaps and reconstructions are performed by those who do plastic surgery. Whenever an orbit is exenterated because of extension from the sinuses or a bare surface is left in the mouth or on the face from the removal of malignant growth, the operator finds it to his advantage to cover this raw surface with skin. It reduces the convalescence several weeks by eliminating an infected, granulating, slowly epithelizing area. By so doing, the surgeon saves his patient from considerable suffering and many unnecessary dressings.

The author mentioned these thin grafts when he stated, "Thin grafts are indicated after excision of a malignancy in order to allow early detection of recurrent or residual neoplasm," and illustrated it with a slide. I fully agree if the area is a nonconspicuous one or if skeletal support has been invaded. If only the excision of a skin lesion is involved, most of our patients prefer a direct closing of the defect, utilizing adjacent flaps as they leave a minimal deformity. The operator can be quite certain that the entire lesion is removed by taking frozen sections as indicated.

A full thickness graft, such as Dr. Huffman showed on the fifth slide, applied to the nose is a gratifying procedure, and skin taken from behind the ear or the clavicular region gives good cosmetic cover for the nose, lids, temple or even the lips.

The use of a graft larger than one anticipates in correction of an ectropion of a lid or lip is always indicated, because contraction is inevitable and secondary excision is easier and cosmetically better than the insertion of another graft.

It was gratifying to see the insertion of a bad result, that of necrosis. It shows the integrity of the author, for this is a condition that any one of us may see in our practice. This same picture will occur in septic or avascular areas.

It was mentioned that it is embarrassing to have hair growing from the buccal cavity. We had a case at the Massachusetts Eye and Ear Infirmary in which a traumatically stenosed larynx and trachea were reconstructed and lined with grafts. This patient had to come in for haircuts of the larynx when the hair grew to a length which caused coughing.

Skin grafting is now employed in the direct reconstruction of an upper esophagus following the excision of carcinoma. It is used to reconstruct ears and noses. All in all, it plays an important part in the field of otolaryngology.

THE PRIMARY SKIN GRAFT IN EAR SURGERY: THE POSTOPERATIVE CARE OF MASTOID AND FENESTRATION CAVITIES

J. Brown Farrior, M.D.
and
Robert G. Iglesias, M.D.
BY INVITATION
TAMPA, FLORIDA

Microscopically perfect technique is the primary requisite for good ear surgery. The skin graft is not essential to good ear surgery. However, the skin graft will make the convalescence more pleasant to the patient, to the otologist and to the referring physician because it reduces the postoperative care.

The skin graft does not eliminate the necessity for meticulous postoperative care of the mastoid and fenestration cavities, but it reduces this care to a level acceptable to the patient.

The postoperative care of the surgical ear is one of our greatest problems. The purpose of this postoperative care is (1) to obtain a dry and a healed cavity in the shortest possible time, and (2) to rehabilitate the patient trained in the care of the ear and relieved of otogenic anxieties. The postoperative care begins with the primary split-thickness skin graft and ends with the rehabilitated patient.

My interest in the skin graft started when I began otology here in New York seventeen years ago. As I recall, this interest started with a lecture by Dr. Morriset Smith at the New York Eye and Ear Infirmary. Through these seventeen years I have explored hundreds of variations in technique, using almost every known method of skin grafting. The skin graft technique which I present today has evolved through these years and from a practical experience in hundreds of operations (more than 750 having been performed since the war). This technique has eliminated many problems, and I hope that each one of you will find some one point which is of value to you, either in your surgery or in the office treatment of these postoperative cases. To me, skin grafting is a worth-while adjunct to Lempert's principles of modern ear surgery.

SURGICAL TECHNIQUE

The primary split-thickness skin graft is taken prior to the ear surgery. The skin graft is preserved by a blood-soaked or saline sponge during the period of the ear operation. Backed with surgical rayon, the skin graft is cut into a pattern and used to line the entire ear cavity at the completion of the operation. The skin graft is held in position from seven to ten days with a gently expanding dressing. The unused remnants of the skin are preserved in saline in the icebox and may be used as a secondary skin graft ten to twenty days later, should denuded areas exist at the time of removal of the packing. Meticulous asepsis is carried out until the end of the third postoperative week.

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FIG. 1—The donor site on the thigh is prepared before the ear and the primary skin graft taken before the ear surgery is begun. This preliminary cutting of the skin graft eliminates the necessity for a secondary surgical setup, permitting a continuous surgical procedure.

Preparation

The upper outer surface of the thigh and of the ear are given an orthopedic preparation the night before surgery. In fenestration cases, great care is taken not to traumatize the external auditory canal and thus irritate the flap. In the operating room the thigh is surgically prepared and draped first, the ear second. This primary preparation of the leg (fig. 1) and taking of the skin graft eliminates the necessity of the cumbersome dual sterile setup and results in a continuous surgical procedure.

Taking the Graft

In recent months we have used the Brown electric dermatome to advantage. Its great advantage is that its use requires minimal training and skill. The calibrations are set at 0.012 inch and the widest maximum graft is taken (3 inches). The graft is taken high on the upper lateral surface of the thigh as near as possible to the anterior superior iliac spine. Although the remaining healed blemish of the skin is negligible, even this is covered with a scanty bathing suit. An additional advantage of the dermatome is that it leaves a smoother donor site.

The dermatome is held with one hand and traction is placed behind it with the opposite hand. When one centimeter of graft has been cut, the corners of the graft are grasped with hemostats and elevated in order to prevent curling of the graft. When the desired amount of graft is taken, the dermatome is elevated, cutting the graft from the skin.

We have taken the greater percentage of our skin grafts with the free knife, using the disposable Weck sexto razor blade (2-inch) and traction blocks manufactured for us by Storz. These traction blocks provide a flat elevated donor site. The free graft is taken with gentle to-and-fro motions from the upper lateral aspect of the thigh. I personally prefer to use the free knife; it is simpler, inexpensive, and saves time. However, this technique requires considerable practice and does not always provide a uniform donor site.

The Pattern of the Graft

The graft is then placed back down on surgical rayon. This surgical rayon has been previously stretched tight over the bottom of a five-inch surgical basin and lubricated with polysporin ointment. The lubricated rayon prevents the curling and shifting which is aggravating in the application of a free graft, as shown by Guilford.[3]

During the course of the ear surgery, the graft and the rayon backing are cut in patterns to fit the individual ear cavity.

The graft and the rayon backing are picked up with tissue forceps and are easily manipulated into position.

The pattern of the skin graft in modified radical mastoidectomies and in fenestration operations consists of two strips of graft (fig. 2). The first strip of graft (1) is roughly oblong, measures 5 cm. long and 2.5 cm. and 3 cm.

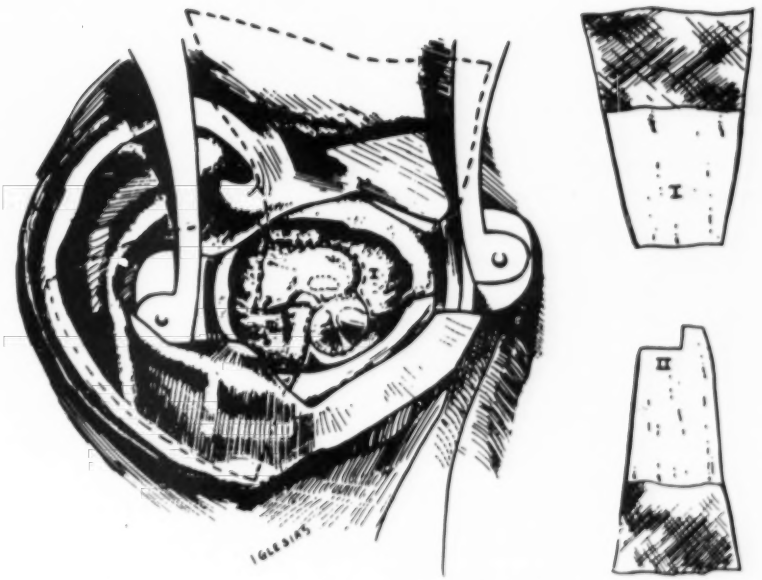

FIG. 2—In the fenestration operation and the modified radical mastoidectomy, the pattern of the graft is cut to cover completely all denuded areas. Strip I covers the tegmen and denuded anterior canal wall. Strip II covers the sigmoid sinus area and bowl of the mastoid up to the flap and skin of the auditory canal.

wide on the ends; this strip is used to cover the denuded portion of the anterior superior wall of the external auditory canal and the tegmen back to the sinodural angle. This graft should come externally to the skin surface to line the enlarged external auditory canal and prevent stenosis. The second strip of graft (II) measures 2.5 cm. by 4 to 5 cm. and is used to line the posterior inferior surface of the cavity. The end of this graft is notched to surround and to approximate intimately the margins of the flap. Every effort is made to cover completely all denuded areas.

Early in the development of our skin graft technique we used small patch grafts, from these islands the remainder of the cavity was supposed to heal. Healing would occur in time, but convalescence has been further shortened by the application of the complete split-thickness skin graft. Cutting of these patterns is done by the surgical nurse during the course of the ear surgery.

In the complete radical mastoidectomy, we usually use three strips of graft (fig. 3). The first two (I and II) are similar to those already described except that the second piece is not notched. The third, and smallest, piece of graft (III) is used to line the middle ear, the denuded hypotympanum, and the denuded floor of the external auditory canal. For this third portion of the graft, we usually select the thinnest margin of the graft. This section is usually 1 to 1.5 cm. wide and 2 to 2.5 cm. long. The graft covers the floor of the canal and extends upward over the medial wall of the middle ear. The upper margin of the graft is placed adja-

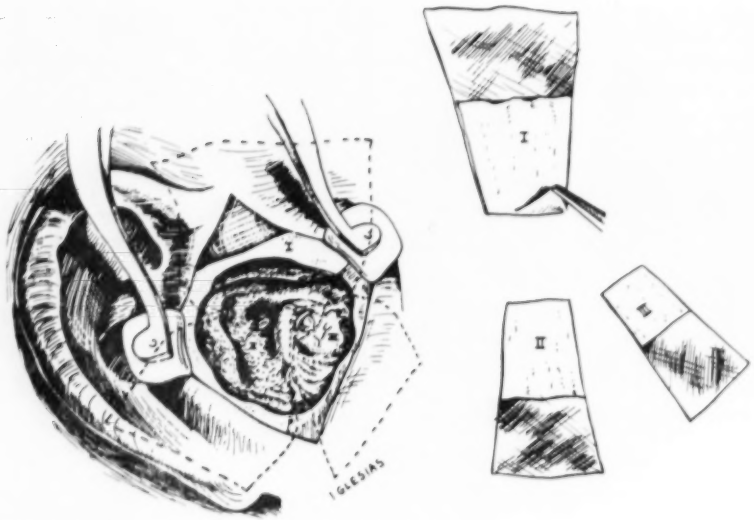

FIG. 3—In the radical mastoidectomy, the graft pattern is in three strips. Strip I covers the medial wall of the attic, the anterior canal wall, and the tegmen. Strip II covers the sigmoid sinus and the floor of the mastoid cavity. Strip III covers the medial wall of the middle ear and the hypotympanum covering all denuded areas.

cent to but never over the stapes. The first portion of the graft (I) is similarly placed down over the horizontal semicircular canal and facial nerve but never over the stapes. When the radical cavity is completely lined with the split-thickness skin graft, a strip of synthetic sponge, 0.5 cm. square and 2 cm. long, is then curled in horseshoe fashion from the hypotympanum upward and around the stapes over the medial wall of the attic. In this way, no pressure is applied to the stapes itself. The remainder of the cavity is then lined with four or five strips of synthetic sponge which have been lubricated with polysporin ointment. The vertical incision in the external auditory canal is sutured and the external flap of our modified Shambaugh incision is tucked inward and held in position by the synthetic sponge packing.

The Leg Dressing

The donor site on the leg is covered first with a layer of surgical rayon which has been lubricated with polysporin ointment. This surgical rayon is so woven that it permits the absorption of exudate but does not permit the ingrowth of fibroblasts. The donor site, therefore, heals under the rayon. A gentle pressure dressing is then applied and held in position with 6-inch strips of elastoplast bandage. Unless there is some evidence of infection, this dressing is left in position for two weeks, by which time the donor site is healed.

POSTOPERATIVE CARE

The postoperative care of the mastoid and fenestration cavities may be divided into early and late periods. The early postoperative care covers the first

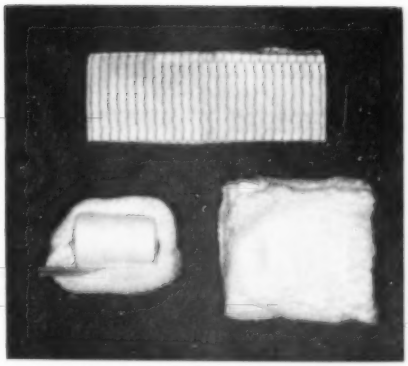

FIG. 4—Rigid asepsis is the primary principle of the postoperative care. Autoclaved ear dressing packages aid in maintaining asepsis. These dressings contain a Kerlix gauze "donut" to surround the ear, a padded Kerlix gauze square, a 2-inch bandage, four cotton applicators to clean the canal and a paper towel to drape the ear. The Kerlix gauze "donut" is made by wrapping a strip of this soft gauze one and one-half times around the 2-inch bandage. The 4 x 4 Kerlix gauze square is padded with a cotton square. The soft bandage causes little discomfort.

three postoperative weeks. The late postoperative care may be further subdivided into care of the healed cavity, care of the infected healed cavity, and care of the incompletely healed cavity. The basic principle of the early postoperative care is rigid asepsis. The basic principle of the late postoperative care is the complete cleanliness of the cavity itself.

Early Postoperative Care

The early postoperative care is concerned with the problems of the head dressing, the removal of the packing, and the final debridement of the cavity. As rigid asepsis is the primary principle of the early postoperative care, we maintain a head dressing for about twenty postoperative days (fig. 4). This head dressing is changed about three times a week with thorough aseptic cleansing of the outer ear and ear canal.

The removal of the packing varies with mastoid and fenestration surgery. In mastoid surgery all packing is removed by the tenth postoperative day. The polysporin or other antibiotic ointments on the synthetic sponge will prevent odor and discomfort in most cases. We have varied the time for removal of the packing from the fifth to the twenty-first postoperative day. If the cavity is infected, the packing malodorous, or there is inflammation of the ear, the packing may be removed as early as the fifth postoperative day and there will still be a good take of the primary skin graft. For a while we tried leaving the packing in for two or three weeks. This was unsatisfactory because the packing would become adherent to the walls, there would be bleeding at the time of removal of the packing, and in some instances the pack, acting as a foreign body, caused infection. Mastoid cases frequently do not remain in the hospital more than a few days. The removal of the soft strips of the synthetic sponge and the surgical rayon is a painless office procedure.

In the fenestration operation, the removal of the packing and the release of pressure on the flap should be so gradual that the patient experiences only the slightest fistula test. If, on the other hand, there is a sudden release of pressure over the flap, there will be secondary edema of the flap and secondary serous labyrinthitis, producing dizziness from a few hours to a day or two and possibly a loss in hearing. To produce the desired gradual release of pressure, the outer packings are removed on about the seventh postoperative day. This allows the inner, coarse packing to expand spontaneously and gradually release pressure on the inner fenestral pack. The inner coarse packing is removed on the ninth postoperative day, leaving only the fine-mesh rayon-wrapped pack over the fenestra. This fenestral pack is removed on the eleventh or twelfth postoperative day. If in the removal of these

packings we produce more than a momentary labyrinthine response, we feel that we have committed a technical error. In fenestration cases, most patients leave the hospital on the fourth to seventh postoperative day, and the removal of the packing is again an office procedure.

After removal of the packing, the next two or three dressings consist only of cleansing the ear canal with the autoclaved cotton applicators, sponging the ear with alcohol and re-applying the head dressing. This is to allow time for exfoliation of the outer layer of the skin graft. This takes about three weeks. For a while we tried to speed up this process and to have these patients ready to return to the care of the referring otolaryngologist by the end of the second postoperative week. This can be done sometimes; but, as a routine, it is better to keep the patient until near the twenty-first postoperative day.

The final debridement of the mastoid and fenestration cavities is begun on the twentieth postoperative day. After thorough cleansing of the ear and canal with alcohol, all eipthelial debris is aspirated from the mastoid and fenestration cavity. The entire cavity is then packed with one-inch gauze saturated with polysporin ointment. This packing is held in position for twenty-four hours with a dual purpose: (1) to lubricate the cavity and facilitate the final removal of residual epithelial debris and (2) to protect the cavity so that the patient may have the hair shampooed, and cut or set. On the twenty-first and twenty-second postoperative days the remnants of epithelial debris are removed by aspiration and audiograms are taken. At the end of this period, three weeks after the operation, the great majority of the cavities are sterile and almost completely healed. If there is a suggestion of infection, we use an appropriate antibiotic.

Late Postoperative Care

At the end of the first three postoperative weeks, the patient is returned to the care of the referring otolaryngologist. The patients are advised to report for examination once a week for three weeks, once a month for three months, and three times a year thereafter. This is the maximum frequency of visits which is acceptable to the patient. The purpose of these visits is prophylaxis. If these cavities are kept clean, they cause little difficulty. If, however, epithelial debris is allowed to accumulate, external otitis will develop. Many of these patients can go for years without visiting a doctor and the cavities will be perfectly healed, but prophylactic cleansing is the best policy. Cleansing is the basic principle of the late postoperative care.

Late postoperative care of the healed cavity. The late postoperative care of the healed cavity consists of removing the epithelial debris and cerumen from the walls of the mastoid and fenestration cavity.

Many physicians are prone to overlook the crusts on the posterior and superior walls. We have evolved the following routine for cleansing these ear cavities. After the preliminary inspection of the cavity, we begin by elevating the crusts from the posterior wall of the cavity. To do this, the speculum and the handle of the ear applicator are almost parallel to the patient's cheek looking in the direction of the occiput (tragoccipital position). The epithelial crust is then elevated from without inward down to the bowl of the mastoid antrum. Secondly, the tegmen is cleaned. To do this, the patient's head is tilted directly away from the physician and a large ear speculum is inserted into the ear canal with the line of vision directly toward the patient's vertex (concho-vertical position). With the ear treatment curette,

the epithelial crust is elevated from without inward down to the medial wall of the attic. The epithelial crusts from the posterior and superior walls are now accumulated in the region of the mastoid antrum. These are grasped with the aural dressing forceps and gradually peeled away from the region of the fenestra. If the fenestral area contains a heavy crust, this is lubricated before it is removed. After the removal of this epithelial cast of the cavity, the cavity may be irrigated with 2 oz. of 1 per cent acetic acid in 70 per cent alcohol. If epithelial detritus remains, the cavity is then packed with vaseline gauze for twenty-four hours. This vaseline packing will lubricate the walls of the cavity, and when the patient removes the packing at the end of twenty-four hours, the epithelial shreds will come away with the packing, leaving a perfectly clean cavity.

Late postoperative care of the infected healed cavity. In the South, we once had the problem of the "carpetbagger," today we have the Pseudomonas infections, and the problem of external otitis. Kept clean, healed cavities cause little trouble. If, however, epithelial debris, cerumen and the like, are allowed to accumulate in the cavity, they may cause any one of the external otitides.

Epithelial detritus forms the background for superficial infection. When more epithelial debris accumulates under the epithelial crust, this detritus simulates cholesteatoma. This may occur in both grafted and nongrafted cases, but it is more likely to occur if there is a small area of residual infection. As this accumulation of debris is not encapsulated and does not tend to erode bone, in my opinion it should not be called cholesteatoma, as described by Peterson.[5]

It is difficult to make a statistically accurate statement on the postoperative care required relatively by the healed grafted and nongrafted cavities. It is my opinion that large cavities with pockets and crevices require more care than small smooth cavities.

In the original operation, exenterating all accessible mastoid cells and applying no skin graft, the resulting large cavity certainly requires more care than does the present-day small grafted cavity. If the healed grafted and nongrafted cavities are of equal size, there is little difference in the long term postoperative care or in their predisposition toward infection.

When infection does occur in the healed cavity, our policy is (1) to cleanse the cavity, (2) to leave the cavity with an acid medium, (3) to apply specific medication, usually in the form of packing, and (4) to teach the patient how to care for the ear. If the patient comes to the office, we first cleanse the cavity as described previously and in addition use suction to aspirate the exudate. The walls of the cavity are then scrubbed with a cotton applicator, saturated with acid-alcohol. The cavity is then thoroughly irrigated with 1 to 4 ounces of 1 per cent acetic acid in 70 per cent alcohol (ethyl or isopropyl rubbing alcohol). At the same time, we instruct the patient in the technique of this irrigation.

In irrigating the fenestration cavity, the tragus is displaced forward, the tip of the rubber bulb irrigator is then directed upward and backward into the mastoid cavity. When the tip of the rubber bulb becomes snug in the ear canal, it is withdrawn slightly to permit the return flow of the solution. Using 1 to 4 oz. of acid-alcohol solution at body temperature, the cavity is irrigated with some force to remove debris.

It has always seemed absurd to me to have the patient instill medication into a

cavity which is filled with infected foreign material. For this reason, we have developed the above technique of instructing the patients to care for and cleanse their own cavities. If there is an acute infection, we use aqueous acetic acid solution, 1 to 2 per cent. When the acute phase of the inflammation subsides, we use 1 per cent acetic acid in 70 per cent alcohol, and in a long-term project where large amounts of irrigation are required, we have the patient use three parts of rubbing alcohol to one part of distilled white vinegar. The patient is instructed to rinse the cavity two or three times a day for three days, daily for three days, and in recurrent cases once a week thereafter. This routine alone will take care of most infected healed cavities and the continued weekly rinse of the cavity (after each shampoo) prevents the accumulation of detritus and re-infection.

In severely infected cases, specific topical medication may be applied. This is applied after irrigation in a clean dried cavity. The specific medication may be applied with or without packing. Medication dispensed for home use is to be applied ten minutes after the ear rinse. Only rarely is there an indication for systemic medication.

Late postoperative care of the incompletely healed cavity. If, after three weeks, the fenestration cavity has not completely healed, there will necessarily be some discharge and these cavities are prone to become infected, particularly with the Pseudomonas bacillus. Such secondary infection may temporarily retard or permanently prevent complete healing of the cavity. In these cases, at the end of the third postoperative week we begin prophylactic irrigations of the ear with acid-alcohol. These patients are discharged and instructed to irrigate the ear two or three times each day as long as there is otorrhea. When the discharge stops, irrigations are continued daily for one week and at weekly intervals thereafter. These ears are usually healed and dry in five to six weeks.

A small percentage of cases will show persistent discharge. We usually treat these with irrigations followed by antibiotics. Occasionally we will use the specific medication on packing. We have almost discontinued the use of dusting powders except when there are infected air cells. I want to warn against the prolonged use of an antibiotic, for this may result in the overgrowth of a secondary organism.

Time was when prolonged otorrhea and excessive granulation were almost the rule in fenestration surgery. I have spent many bloody hours curretting these granulations and using all types of treatment from gentian violet to the antibiotics. These messy ears are now almost extinct. However, a small percentage of cases show persistent discharge in spite of all treatment; this usually results from infected residual mastoid air cells. In these cases I advise revision of the mastoid portion of the surgery and new application of a skin graft. This has been necessary in only 4 of more than 500 cases. There are two or three others which should have this secondary skin graft. The time for advising such a revision of the mastoidectomy must be left to the judgment of the individual surgeon, though I am beginning to believe that this mastoidectomy should be done in those treated cases which are not healed by the sixth postoperative month.

SPECIAL USES OF THE SKIN GRAFT

1. *Facial Nerve Surgery*

In applying a facial nerve graft, we first simulate the nerve graft by placing an adrenalin-soaked string of an appropriate size in the prepared fallopian

canal. The medial wall of the cavity is then lined with a single piece of split-thickness skin graft and the middle ear packing inserted. The skin graft is then elevated from behind forward and the adrenalin-soaked string replaced by the facial nerve graft itself. Microscopically accurate, maintained, end-to-end anastamosis is ensured by immediately covering the facial nerve graft with the split-thickness skin. Healing is facilitated.

2. *The Skin Graft as the Fenestration Flap*

Dr. George Shambaugh,[8] the Doctors Antoli-Candela,[1] and others have used the skin graft as a substitute for the tympanomeatal flap in fenestration surgery. I have 4 such cases. Certainly the skin graft will work as a flap to cover the fenestra, and it may have some advantages. If for any reason I do not like the appearance of the flap, I substitute a split-thickness skin graft.

3. *Stenosis of the External Auditory Canal*

In congenital and acquired atresia of the external auditory canal the skin graft is indispensable. In congenital atresia, the skin graft may be used to cover the fenestra.[4,6,7,9]

4. *Dural Exposures*

In uncomplicated mastoid and fenestration surgery, debrided dural exposures may be covered directly with a split-thickness skin graft. In such cases, the exposure is inspected under magnification and bone spicules are removed. In innumerable cases, we have covered such dural and sinus exposures without complications. The absence of complications is attributed partly to the accurate debridement and partly to the postoperative use of antibiotics.

5. *Septic Complications*

In septic complications of the chronically infected ear a partial skin graft is indicated. If there is an epidural abscess, a brain abscess, or a sigmoid sinus thrombosis, we do not place a skin graft over the involved dura or sigmoid sinus. In the rare labyrinthotomy or petrus apicotomy, we do not cover the drainage area with a skin graft. In our opinion, the skin graft in the remainder of the cavity not only facilitates healing but also permits better postoperative care and possibly prevents additional complications. In such cases we may insert a drainage tube down through the packing to the involved area.

6. *The Eustachian Tube*

The primary requisite for the prevention of the "tubal ear" following mastoid surgery is the complete and accurate removal of the hypotympanic cells without producing stenosis of the eustachian tube at the isthmus.[2]

In the course of the radical mastoidectomy we routinely cover the medial wall of the middle ear and the orifice of the eustachian tube with the primary skin graft. However, any success which we may enjoy in preventing these "tubal ears" we attribute not to the graft but to the accurate bone surgery.

SWIMMING

For years swimming has empirically been prohibited after surgery of the ear. I see no reason to deny the pleasure of swimming to patients with healed cavities following modified radical mastoidectomies, and fenestration operations. I permit my patients to swim. My patients with skin grafts usually resume swimming within a few months after surgery.

Patients are instructed to fill the ear canal with cotton before swimming and

to wear a bathing cap. This is done not with the fear that water will do damage but to prevent labyrinthine stimulation from the sudden inrush of cold water. Many patients go swimming without these precautions. These patients are instructed to do ordinary surface swimming and surface diving but are warned against the possibility of labyrinthine stimulation from high or deep diving. After swimming, the patient rinses the ear cavity with acid-alcohol solution to remove moisture and leave an acid medium. No problems have developed among the many, many patients who do ordinary swimming and water skiing, however I have some concern about one patient who insists on diving to greater depths in spear fishing.

COMMENTS

The skin graft has been of great value in the radical mastoidectomy and in fenestration surgery. In usual modified radical mastoidectomies, the cavity is small so that healing is not a great problem.

In the radical mastoidectomy, the goal of the postoperative care is to obtain a smooth cavity which resembles the contour of the operated bone. The skin graft expedites this goal. The skin graft helps to prevent the cavity from filling up with granulations and narrowing the canal, all of which might result in residual disease being trapped behind the stenosis.

In our 527 fenestration operations we have gradually changed our technique from the complete exenteration of all accessible mastoid air cells with no skin graft to the present-day partial exenteration with the complete primary split-thickness skin graft. These improvements have greatly reduced postoperative care to the joy of the patient and the surgeon. Our greatest postoperative healing problem occurred in the early cases with the original endaural incision and complete exenteration of the mastoid air cells. These cases took many, many months to heal with the problems of discharge, excess granulations and bleeding. These were sloppy ears and, in spite of the gratifying restoration of hearing, jeopardized fenestration surgery. The convalescence was improved some by preserving the external conchal-canal flap. We have used the skin graft in 376 operations with gradually improving technique and results.

In my opinion, the complete skin graft is more essential in fenestration surgery than in other varieties of ear surgery because it seals, preventing a secondary infection in the residual mastoid air cells. The five or six retention cysts which I have seen in these peripheral cells have all developed in cases which were not skin grafted. By and large, the smaller the mastoid the more rapid the healing; on the other hand, very large pneumatic mastoids are more likely to cause postoperative difficulty.

The modified radical mastoidectomy is usually performed for a cholesteatoma in a relatively small sclerotic mastoid, in which healing is no great problem. Occasionally a modified radical is indicated in a pneumatic mastoid where there is secondary cholesteatoma in the antrum and attic. In these cases we remove and ablate the mastoid tip and apply the primary skin graft only over the tegmen. Secondarily, a refrigerated autogenous graft is applied two weeks later to the ablated tip. This results in a smaller cavity which is more easily kept clean.

At one time, we used the secondary skin graft now advocated by Guilford. Secondary grafting has some points in its favor. In my opinion, these are outweighed by the single complete surgical procedure of the primary graft.

SUMMARY

1. The technique of skin grafting here presented has evolved from practical experience of more than 750 ear operations since the last war, the total experience extending over a period of seventeen years in otology.
2. The detailed technique of taking and applying the graft is described.
3. The early postoperative dressings are described.
4. The late postoperative care is described for the healed cavity and the incompletely healed cavity.
5. The special use of the skin graft in otological complications is outlined.
6. The patient's instructions for the care of the postoperative cavity are described.
7. Postoperative cases with the completely epithethialized cavities are permitted to swim.
8. The complete primary split-thickness skin graft has greatly reduced the problems attendant to the postoperative care of the surgical ear.

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DISCUSSION

BEN H. SENTURIA, M.D., St. Louis, Mo.: I should like to congratulate Dr. Farrior on a clear and concise presentation of his extensive experience in the use of the primary skin graft in ear surgery. In particular I would like to commend him on the straightforward manner in which he has described not only the postoperative treatment of his successful cases but also the method by which he treated those which did not respond promptly. It is of utmost value to hear a frank discussion of the unsuccessful cases—that is, the treatment of the infected, unhealed postoperative ear—since, needless to say, all of us who do mastoid or fenestration surgery have one or more intractable postoperative draining cases.

I have heard various explanations for these persistent otorrheas. In some places I hear that these cases are due to incomplete surgery and to open eustachian tubes. In others, it is ascribed to allergies, and most often it is blamed on superimposed infections. Some or all of these theories are undoubtedly correct, but I should like to call your attention to several factors which may have been overlooked.

In our research dealing with the skin of the external auditory canal, sponsored by the Air Forces and the St. Louis Otological Foundation, we have been impressed with the remarkable ability of normal intact skin of the ear canal to maintain itself in a healthy state. Only a relatively small number of endogenous bacteria, primarily the staphylococci and some micrococci, will grow on normal skin.

If, in one manner or another, we alter the chemical or histological integrity of this skin, various bacteria, especially the gram-negative bacilli, or fungi will grow luxuriantly. I shall refer to these alterations briefly.

Not too many years ago, in the St. Louis area it was rare to hear of an otologist who used skin grafts to line the mastoid cavity. The postoperative healing, as Dr. Farrior indicated, was therefore agonizingly slow. Yet, when complete healing occurred, one rarely saw crusted, infected, ulcerated epithelial lin-

ing which broke down with the first mild U.R.I. One has difficulty recalling many cases of diffuse otomycosis requiring every therapeutic trick in order to obtain a dry ear.

Is it possible that the postoperative draining ear is due to some inherent deficiency in the primary skin graft technique which is not present when the remaining skin of the ear canal is allowed to regenerate and the intervening gap is filled with scar tissue? (Slide 1) I should like to show you the normal skin of the ear canal which reveals numerous sebaceous and apocrine glands (slide 2) and a split-thickness graft from the medial surface of the thigh. (slide 3) It is no wonder that this crippled split-thickness skin exfoliates layer after layer of keratinized epithelium and is poorly resistant to water and infection. Even if the split-thickness graft takes well and heals, it is not surprising that this fibrous wallpaper shows little resistance to infection, ulcerates and develops diffuse granular islands over the tegmen and in the mastoid cavity. Thus, it is not unexpected that many of us have come to the same conclusion as Dr. Farrior and advise our patients to use some form of home irrigation. We prefer the use of aluminum acetate irrigations, but in view of the difficulties in eliminating all the water from the depths of the mastoid cavity, the acetic acid-alcohol mixture described by Dr. Farrior offers distinct advantages.

In closing, I should like to present three thoughts in reference to the care of the postoperative mastoid cavity. First, my associate, Miss Alford, and I will report shortly the very excellent results obtained in the use of an aqueous suspension of neomycin and hydrocortisone acetate in the treatment of the persistently draining postoperative mastoid and fenestration cavities. Ears that have resisted all forms of therapy for years, even those with granular walls from which the Pseudomonas or Proteus were cultured, have responded in a matter of days. Our results have been most gratifying and I hope that these findings will be confirmed by others.

Second, we have repeatedly urged the use of full-thickness primary grafts in the mastoid cavity, particularly in the region of the tegmen. If we can reapply normally functioning, healthy skin with intact dermis containing apocrine and sebaceous glands, we can hope for much better postoperative results. Recently, Dr. Walsh used full-thickness axillary skin grafts in one series of cases and full thickness postauricular skin grafts in another. He informs me that some problems arose in dealing with the donor sites but that very satisfactory healing occurred.

Finally, I wish to re-emphasize the important role played by the skin of the outer half of the external auditory canal, particularly the superior and inferior walls. I should like to point out the irretrievable damage caused by cutting away large segments of skin in this area and to condemn the practice of trimming away the subcutaneous tissue with the contained apocrine and sebaceous glands.

Needless to say, I am sure that as a consequence of Dr. Farrior's paper all of us will have a clearer understanding of the problems of skin grafting in ear surgery.

GEORGE SHAMBAUGH, M.D., Chicago, Ill.: Dr. Farrior has asked me to mention our experiences with skin grafting in fenestration surgery. For the past year, in about 300 operations we have routinely Thiersch-grafted the fenestration cavity at the time of surgery. It is true that in some cases the cavity will be completely epidermized and dry as early as four weeks after operation, but in a good many cases, the initial healing is followed by a breakdown in one or more areas and as long a period of healing as is seen in cases not grafted. Moreover, the finally healed cavity seems to us to be less desirable after primary skin grafting in that there is an increased amount of troublesome desquamation, the walls of the cavity are more irregular and therefore more difficult to clean, and the cavity is larger. Accordingly, we have now gone back and no longer do primary skin grafting of our cavities. With scrupulous aseptic technique in the postoperative care, a great majority of these cavities heal very nicely by themselves with a healthier appearing skin and a smoother surface when the epidermis grows in by itself over a thin pad of granulation tissue.

I should also like to mention our results with the technique of covering the fenestra itself with a split-thickness free Thiersch graft rather than the usual pedicle flap. We tried this technique in a series of 76 consecutive operations in the hope that postoperative bleeding and exudation into the labyrinth from the periosteal surface of the flap might be avoided, thus eliminating one of the causes for failure due to serous labyrinthitis. This hope was not realized. When the postoperative hearing results were analyzed in this series of cases it was found to be essentially the same as in a similar group of cases operated upon by the usual pedicle flap technique, both in the proportion of cases receiving an improvement and in the average number of decibels gained. Accordingly, we have again gone back to the pedicle flap technique.

A PLEA FOR THE BEST POSSIBLE PREINDUCTION PHYSICAL EXAMINATION

FORREST J. PINKERTON, M.D.
HONOLULU, HAWAII

SOME of my listeners will hold that a discussion of this kind is out of place at this meeting. They will contend that those in attendance want to hear scientific discussions only. Others are opposed to listening to a discussion of subjects of vital importance to the welfare of the country and the whole people, no matter what the nature of that discussion may be. Too many in our profession are ignorant of, and totally disinterested in, the affairs of State, the Nation and the World—being entirely satisfied if they can develop a new procedure, a new instrument or a new syndrome, and have it bear their name. These days it seems to me equally important that the medical profession consider well the affairs of a political, economical and moral nature.

Those who have had the honor of military service will be interested in my remarks, and I hope those of the younger group who will soon be selected may find something in my words that will assist them in their service assignments.

Some of you may recall having seen press comments and references to a report I made in May on one phase of this subject. The many clippings and communications sent to me seem to indicate wide public interest in the subject. While I was correctly quoted in those press comments, too much emphasis was placed on one or two of my statements, without a word of explanation as to how I arrived at my conclusions. A bald statement taken out of context may imply something quite different and may be magnified beyond its importance.

Having served through World War I and for some time thereafter, I had wide experience with careless, indifferent and inaccurate medical examinations, plus absent or grossly inaccurate physical records—both pre-induction and at service separation.

Between World Wars I and II, I continued close contact with the services, not only as a professional consultant but in other capacities. During World War II, I served as chairman of Procurement and Assignment for Hawaii and as Director of Blood Bank Operations, and held other professional assignments. Since World War II, I have continued as consultant and hold certain other highly confidential positions. In February-March 1953, I was commissioned to make a critical survey in the Far East zone of combat, on all matters pertaining to oto-ophthalmology and allied services. The war was then at its peak. It was one of my duties to maintain closest contact with those high and low in the armed service, and it was my experience and observations in Korea that confirmed many of my following statements.

The above is stated for the sole purpose of establishing my background of experience and training that you may accept me as qualified to make certain basic observations, criticisms and recommendations.

Primarily I must make it crystal clear that I firmly believe that every young

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man and woman should serve in the armed services in some capacity. Such service makes better citizens, inspires a better understanding of citizenship and loyalty and imbues self respect and self esteem in contrast to those who have denied or have been denied their patriotic duty.

Regulations with loopholes that make it possible for a reluctant inductee to exaggerate a perfectly transparent or trivial defect for the sole purpose of evading his patriotic duty must be totally condemned. A history of a headache, a tag of hemorrhoids or a religion of devout cowardice is hardly enough excuse to escape one's duty to his country. Such individuals need the benefits of military service to bolster and strengthen their moral fiber and their Americanism.

The time has long since passed when the Armed Forces can maintain their manpower physical standards any differently than does any large enterprise. There is a place in the Armed Services for most physical defectives, now gainfully employed in industry. Careless induction techniques with casual and indifferent physical examinations, grossly inaccurate, deficient, or absent records of physical defects, must be considered as one of our greatest national disasters that could have been prevented, and can be prevented in the future, by the simple application of meticulous accuracy and care in our pre-induction physical examination, and the accurate recording thereof.

Selective Service is mandated to induct, and is given the responsibility and power to make and enforce regulations concerning all phases of induction.

It seems to the minds of all of those with whom I have discussed this problem that the physical standards now required for military service should be revised downward with the intended goal of inducting into military service a fair share of that great army of physical defectives and mental reluctants. These many draft escapees could easily be classified and placed in safe and secure positions, safe for themselves but, more particularly, safe for their country. All this could be done by a determined effort on the part of the administrative heads of the various armed units. But, I repeat again, when they are inducted into the service they should have the benefit of a physical examination, the very best and probably the only examination they have ever had; and what is equally important, every detail of that expertly done examination should be placed on their record. Failure to record at all, or inaccurate recording, is the basis of most of my criticism.

I have consulted many in authority within the services but it is not within my province, power or desire to suggest where the responsibility lies in the correction of the things I have mentioned.

We can depend upon the statistics that say every third man is unfit mentally or physically. Statistics also tell us that one out of those three men who passed as *physically* qualified did not serve because he was either too dumb or too smart—the odds are in favor of the smart ones.

Evacuation of the vast majority of the wounded from Korea was done by air through Honolulu. All of them were admitted to Tripler Hospital for 24 to 48 hours except those that were held longer for definitive care because of the severity of their wounds and/or complications. Something like 64,600 air evacuees have passed through Honolulu from July 1950 to September 1953; of these, approximately 45,000 had war wounds. Eight and one-tenth per cent of all casualties in Korean World War III were eye injuries, or eye diseases and defects.

My concern was most forcibly aroused in 1952, when a study was made of 500 *consecutive* air evacuee eye cases. This preliminary study revealed that 27 per cent (135) of the 500 were due to gross errors in induction techniques. Of the 135, 13 per cent (17) should not have been returned to the mainland since it seemed clear to us, and other examiners, that they could have done satisfactory duty behind the front lines. The remaining 118 were obviously unfit for any except menial duties because of congenital cataracts, uncorrectable visual defects and other serious eye conditions. These 118 should have been returned, but most important, they should never have been allowed to leave the United States in the first place.

Another group of 2457 consecutive air evacuation eye cases was now studied, 1568 of which were due to actual battle injuries. The Army had 1372, the Marines 168, the Navy 15, and the Air Force 13. In the group of 2457 air evacuees were 889, or 36 per cent, being returned for eye defects totally unrelated to war injuries or service connected, directly or indirectly; a minor percentage were activated old eye lesions. Just think this over carefully—36 per cent of this one series of 2457 consecutive eye cases were congenital or pre-induction conditions such as old injuries, congenital malformations, corneal scars, old diseases now healed, uncorrectable high refractive errors, very high myopia, the trophias, advanced retinitis pigmentosa, congenital cataracts, and numerous other conditions, including far too many pure eye malingerers. Of the 889, 526, or 21 per cent of the total 2457, were grossly and obviously unfit for overseas duty.

Most of these 889 evacuees had done no duty of any kind in the Far Eastern theatre, but spent from a few weeks to a few months going from center to center, unit to unit, specialist to specialist, seeking one objective only—a transfer back home because of a partially blind eye which they knowingly had always had, or any one of many other congenital defects of which they had always been aware. For the first time in their lives they became concerned because one eye was crossed, one lid drooped, or one ear was deaf. Circumstances suddenly caused them to focus their attention upon their well-known (to themselves) defect. Now they insisted that these defects, which they had always had and which never caused them trouble before, suddenly were so troublesome that they honestly, but more often dishonestly, were unable to perform any assignment, not even K.P. duty. So they started on their rounds from doctor to doctor until finally they accomplished their objective—a transfer back to the States. This technique works, because 258 (10.5 per cent) of the 2457 consecutive eye evacuees accomplished their purpose.

The type of individuals which I have just described constituted most of the hundreds that I examined while I was in Korea, and my remarks are based upon an experience of being right on the ground in every major medical installation in Korea, Japan and other areas.

At this stage of operations we must recognize that the service man is firmly set and progressing nicely along that well-worn path to becoming discharged, a veteran with a complaint and a candidate for disability, compensation for life for a condition predating his induction, frequently even predating his birth. His induction record, too often silent, too often inaccurate, protects him, and he knows it. Cases are known where preinduction hearing defects were well-recorded in civil life, and after discharge a hearing recheck, repeatedly

done, actually showed the hearing to have improved, yet awards are made for as much as 50 per cent. But this is routinely explained away by a dismissing statement, "the pre-existing or pre-induction defect or condition was aggravated or made worse by reason of military duty." The opinion of the consultant is not solicited. Getting a compensation award is not too difficult for one of anemic principles. First he must develop a fixation on some defect or peculiarity and then stick to it. Once compensation is awarded to these dishonest claimants, we may expect to receive annual applications for an increase in the compensation for any reason that comes first to mind. I know of no instance where a pension was reduced.

Of course there are those controversial cases where the services are jointly responsible for a situation such as the following: The soldier has had a pterygium for many years prior to his service. He complains to his unit medical officer, who performs an operation, with good or bad results. That soldier will get a pension if he tries hard enough. The lesson here is: Don't touch such a condition surgically even though the regulations permit it; send the patient back to a base hospital. That is where he wants to go anyway, he has learned the way out through the experience of his friends. A Board, well selected, should determine all elective surgery. From all the foregoing you may have the idea that our service men are all bad. The truth is far from that, because I can attest to the fact that an overwhelming majority of our boys in the service are the finest fellows one will ever meet. The guilty ones cloud the horizon. If we refer back to my percentage of induction errors for eye defects alone, we find that 661 unfit men were trained for overseas duty at a cost of $10,000 per man (a conservative estimate). This small study of 2457 consecutive eye cases then suggests that most of $6,610,000 of the taxpayers' money was wasted. Of this insignificant number of 661 with nonservice connected eye defects alone, we must assume that many of them will get varying amounts of compensation for the rest of their lives. Computing other causes for pensions percentagewise as compared to eye conditions will reveal a fantastic cost in billions of taxpayers' dollars.

The Armed Services can teach a man how to fight, but they can not teach him the reasons why, nor can they supply him with character and patriotism which should have been ingrained into his very soul by home, family, church, schools and our government. For twenty years prior to our present administration we have suffered a gradual deterioration of our morals, our spirits and our souls. We sorely need to recover our pride, honor, and honesty, not only in our government but for the soul of every American citizen.

Two years for the defense of his country out of the life of a young man under 25 could and should be his richest experience. But sometimes I get the impression that people today are almost ashamed that their son or daughter is in the Service.

We can not blame the Armed Forces for the flimsy characters whom they inherit through the draft. Every draftee carries with him into the services the influence of his immediate family, relatives and friends. That influence may be the good patriotic American way of life, or it may be one of little or no respect for the uniform and flag. Efforts to escape the draft and military duty are deadly routine by the latter.

Now let us look at some more statistics. In Korean World War III, 108,650 men were wounded. As of August 1 this year, about 114,000 pensions

or compensation claims had been granted. The Truce on July 27, 1953, abruptly stopped the wounding, yet the application for pensions or compensations continues on at the rate of about 5,000 per month.

Pensions or compensations granted to date for Korean veterans alone exceed the number of those wounded by many thousands, and it is to this group I refer as having gotten pensions for conditions having no connection with their service. It is computed that by the end of this year we will be paying pensions to 130,000 Korean veterans. Why are we being taxed to death? Explore as I have and you will find the answer. We will soon be paying pensions to three million veterans of World Wars I, II and III. About 21 million veterans are with us today. This number is increasing at about one million per year. Computing percentages we find that 14 per cent of all our veterans are getting pensions and/or compensations.

All this boils down to a few recommendations:

1. Reduce the physical standards for military duty in all the branches, so that we may keep in balance good physical manpower outside the service as well as in the service. General Hershey says, "Divide the bad all around, everybody take some." Perhaps our deteriorating, physical, mental and moral caliber will be strengthened thereby.

2. Place younger medical officers along with the older men in properly zoned induction centers for periods not to exceed six months. To keep them there longer means deterioration in their work. They will do better work if they know how long that duty will last. Examine every segment of the inductee's body and record every detail on his induction record. This examination may be the first (and perhaps the only) real physical examination he has ever had. At all costs, record the findings, and then in days to come believe and respect the reliability of that record. It will save us billions. Our present "table of organizations and equipment" does not provide for a skilled eye, ear, nose and throat specialist. The "induction team" should be composed of the best trained men, adequately paid, together with a highly qualified secretarial and clerical staff. This staff will conserve the time of the examiners and will ensure accuracy in recording.

3. Go all out for the deserving man and fight to the death in the interests of anyone who, having served his country in war or peace, is not properly and fully compensated for injuries or disabilities in connection with that service. But such inequities are so rare and infrequent that one sometimes doubts their existence. Professionals work in their connivances to get more and more for those who have earned it less and less. We should refuse to support any veteran whose pension claims are definitely not of service connection, and likewise we should not aid and abet an ex-service man in degrading himself by signing the "pauper's oath" just to get free hospital and medical care for non-service connected illnesses, unless he is in truth penniless and destitute. Are these thousands and thousands of veterans really penniless? Or is it something more tragic? Such as the lack or loss of personal pride, courage and integrity that allows them to succumb to "creeping socialism."

4. We must stop those who gain their political office by fostering dishonest compensation or pension claims. The best men of our profession have served or are serving in the Armed Forces, and are one group with the power to correct the evils I have referred to. We can accomplish that service to our country by doing the best

physical examination possible, by meticulously recording that information, and by standing steadfast in the days to come when these fantastic claims for pensions come to us for approval. Our young men need, more than anything else, a new philosophy — honor, integrity, honesty and pride. Some seek the grand prize of a lifetime—a pension for something they have not done, a service they have not performed. To demand a reward, a prize or a gift for a patriotic duty to their country is unthinkable, dishonest, dishonorable and totally un-American.

5. Economically, our nation is practically bankrupt. The largest item of expense to the taxpayer is the Department of Defense, and this appears to be impossible of much reduction. The next biggest cause for our 60 billion dollar budget is the government payroll for more than seven million varied government employees, which the experts tell us can easily be reduced by 30 per cent. This would cut that 23 billion dollar salary budget by 8 billion dollars a year.

Another area of fantastic cost to the taxpayer is the Veterans problem. Our socialists, our professional "bleeding hearts" and political gangsters in our welfare offices instead of teaching and promoting dependency, should be teaching Americanism, which is pride and honor and independence. For 25 years their influence has been filtering down through every level of our people, so it is no wonder that so many of our veterans have lost their pride and soul in seeking an easy way out. It is this group of do-gooders who have, over the years, accused the medical profession of negativism when we refuse to follow the socialistic line by being sympathetic to their great army of "dependents" created by a socialistic philosophy that had its origin high in our government for purely political purposes. Our present administration is great, but it will be greater when it rids itself of those "creeping socialists" and replaces them with those who believe it is wrong to sign a false dependent oath, or who seek pensions by false means and claims. Today these documents are signed untruthfully with no thought of right or wrong, simply because it has been common practice.

CONCLUSION

World history seems to indicate that after each great war, some deterioration takes place in the very souls of the countries involved. With the ease of our American way of life, perhaps our values are more materialistic today, and our instinct to live is not as keen as that of those who have had to struggle for existence. Our best men in the service are those who will never be led into socialism by accepting or seeking a pension for something they have not earned. They are the boys we want to come home from the wars to take the leadership of our country away from those who have been incapable of contributing anything to our spiritual and moral betterment.

The medical profession within and without the Armed Services seems to stand alone against those forces who have done things wrong in the past, and by repeated doing think they are right. The statistician of the present can accurately compute our financial disaster of the near future if we continue on at our present rate of dissipating our economy, our pride, our honor and our independence.

DISCUSSION

BRITTAIN F. PAYNE, M.D., New York City: Due to the fact that Dr. Pinkerton had to return to Hawaii early, his paper was shifted to yesterday and inadvertently I missed it. I am not an official discusser, but due to my

connection with the United States Air Force up to this last July, during which time I have been national civilian consultant, I have traveled over a hundred thousand miles by air to all parts of the world and I have seen the military setup from French Morocco all the way to Formosa and in various parts of the globe.

I am familiar with the type of individual that we have in our Air Force, also in our Army, and the things that Dr. Pinkerton had to say yesterday as to the preinduction examination are absolutely correct. In other words, due to the fact that we do not screen these individuals carefully before they come into the military service, you and I as taxpayers are burdened with an added pension list that could obviously be stopped at the very beginning.

There is one thing that I should like to make clear, and that is that it is not the fault of the doctors in the military service. That goes for the Air Force, for the Navy, and for the Army. After these individuals are inducted into the service they have no choice. They have to accept them, and sometimes it takes months, as you know, to evaluate the person and find out whether or not he is actually qualified for military service. In the meantime, in those months, these people can claim that their disability occurred in military service, and they are "on our necks" for the rest of their lives.

So I want to emphasize the things that Dr. Pinkerton had to say and to try to help in creating an awareness of the situation, which I think we can help a great deal. It goes right back to the original draft board examination. You and I are paying for it. I just hope that we pay some attention to the pre-induction setup and that we do something about it.

It would have been a privilege to have heard Dr. Pinkerton yesterday, because I know what he went through, through the months of February and March in Korea. I followed him in Korea in April, and it is not a very pleasant place to be, even in April, in the spring with cherry blossoms and all that sort of thing. He was there when it was cold and he did his job very well indeed. He didn't make himself too popular with some of the officials in the military service due to the fact that he called the cards as he saw them. However, I think he has done a great thing in bringing to our attention the importance of the pre-induction examination in the military service and, ladies and gentlemen, I hope we do something about it.

In Memoriam

Emmett Daniel Boaz.....Pittsburgh, Pennsylvania
November 6, 1954

Leon Edward Brawner.....St. Simon Island, Georgia
December 4, 1954

Robert Rhodes Chace.....Bronxville, New York
November 24, 1954

Martin Cohen.....New York, New York
October 29, 1954

John Crockett Easton.....Springfield, Ohio
September 30, 1954

Joseph Feldman.....Palm Beach, Florida
October 7, 1954

Harold Lowry Hickey.....Denver, Colorado
December 31, 1954

Gunnar Holmgren.....Stockholm, Sweden
September 6, 1954

Joseph Josiah Hompes.....Lincoln, Nebraska
November 16, 1954

Etta Charlotte Jeancon.....Los Angeles, California
October 30, 1954

Richard Augustine Kearny.....Washington, D. C.
October 4, 1954

Frederick Edwin Knowles, Jr.....Baltimore, Maryland
June 5, 1954

George Edward Lathrop.....Sumner, Illinois
October 12, 1954

Robert John Percival McCulloch....Toronto, Ont., Canada
December 26, 1954

Michael James O'Connor.....Tucson, Arizona
October 3, 1954

Kaspar Pischel.....San Francisco, California
April 9, 1953

Everett Powers.....Carthage, Missouri
December 16, 1954

David Henry Rhodes.....Pittsburgh, Pennsylvania
October 31, 1954

Hunter Watt Scarlett.....Bryn Mawr, Pennsylvania
December 23, 1954

John James Sheedy.....Plainfield, New Jersey
December 7, 1954

A. Edward Sherman.....La Mesa, California
December 16, 1954

Ernest Gerard Smith.....Lancaster, Pennsylvania
November 20, 1954

Albert Conrad Snell.....Rochester, New York
December 11, 1954

Frederick Leaver Stauffer.....Salt Lake City, Utah
November 10, 1954

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THE USE OF FORMAL DIDACTIC TRAINING IN GRADUATE MEDICAL EDUCATION

The usual teaching methods in almost all American institutions of learning, from the primary grade schools to graduate schools in universities, consist of planned courses of instruction in a curriculum that covers a number of subjects considered as desirable fields of knowledge consistent with a learned citizenry and an avocation.

In a small number of teaching and research institutions, usually of highly specialized character, the didactic system of instruction may be minimized or abandoned and the student encouraged to explore for himself the vast field of knowledge and to form his own opinion of the importance and application of new discoveries and their relationship to the present understanding of things as we know them.

The difference between these two systems of education is largely a matter of timing, and both are employed to some degree in all schools. The beginning of education in any known subject is through instruction covering the fundamentals or accepted facts as a base from which independent activity can be extended to the utmost capacity of the student. All knowledge is based on established conceptions formed in past ages and accepted in a common understanding that constitutes our civilization. The cumulative knowledge of our time, which is a matter of record, must be transmitted to new generations by association of teacher and pupil through one or more of the senses of perception—sight, hearing and touch. It is impossible for a human being to learn to talk, read or write without a teacher.

There is no question that mass teaching is mandatory in today's economy. The universities today are faced with the problem of housing campus students in addition to providing classroom and laboratory facilities for an increasing enrollment that is climbing at a faster rate than the rate of increase in population. Even though financial support is obtainable, the problem faced by educators today is how to maintain the present standards of education.

The growth of the student body ne-

Excerpts from a paper read before the Advisory Board for Medical Specialties, February 5, 1955.

cessitates comparable increase in physical plants, including classrooms, auditoriums, libraries, laboratories, gymnasiums, heat, light, power, dormitories, restaurants, transportation of students and maintenance personnel. Then too, the faculty must be increased and the administrative department augmented from "prexy," as all students in former generations have known him, to a president, assistant president, two to five or more vice presidents, assistant vice presidents, registrars, secretaries, press, radio and television directors, housekeepers, superintendents and a host of bookkeepers, clerks and accountants, none of whom belong to the teaching faculty. The over-all cost of public education is not only the cost of hiring teachers for large classes of students. As student campus enrollment is increased in the universities and colleges, there is no alternative to increased spending, which means increased grants and taxes to support the educational system.

What of graduate education in the years to come? The population increase in the United States poses a statistical problem. During every minute of 1953 the United States increased its population by slightly over five people. This amounts to:

300 an hour
7,300 a day
51,000 a week
223,000 a month
2,725,000 a year

In 1954 over 4,000,000 children were born in the United States.

In one Upper Midwest state in the last generation, college enrollment increased three and one half times while the population increase of the state was less than double. A higher proportion of college-age men and women are attending college.

Year	
1900	4.0%
1910	4.6%
1920	7.2%
1930	11.9%
1940	15.4%
1950	20.0% (excluding veterans)

Resident credit students in one large state university showed an increase in graduate-professionals from 26 per cent in 1929-30 to 39 per cent in 1954-55. Instruction costs increase with the advancement of students. For one dollar spent on the education of a freshman, it is necessary to spend two dollars to purchase the same amount of service for a junior or senior, and over four dollars to purchase the same unit of service for a graduate student, or graduate-professional student enrolled in Medicine, Dentistry or Public Health. College enrollment is increasing by waves coincident with world wars, partly due to federal aid in one form or another.

What alternatives do we have to institutional on-campus training in graduate medical education?

A teacher may be an expert in a special subject, for example embryology, one of the basic sciences. As a worker in embryology he becomes well known in the medical field and his lectures are in demand. He can lecture to an assembly of 200, or more, students, and by giving lectures to a number of groups he can easily give instruction to a thousand or more students. As he lectures to graduate students who have some knowledge of the subject, a few hours will serve to stimulate the interest of his listeners in further studies on their own. The cost is spread over a large number, and the cost per student is minimal. In the medical so-called basic sciences, off-campus instruction, as in postgraduate hospitals,

can be provided by teachers whether or not they are professional practitioners or full-time members of a college faculty.

Let's take another example. In a refraction clinic the ranges are set up so that one instructor can work among a group of four to six students, supervising their work on patients. Personal contact between teacher and pupil is essential to good teaching in refraction, and one teacher can properly supervise only a limited number. The cost of one teacher is divided by the number of students he attends. Compared to the lecturer on embryology, the instructor in clinical work is an expensive member of the faculty.

Graduate study has become of such importance in the field of clinical medicine that medical schools and teaching hospitals have established staffs and facilities to provide for residents who wish to extend their clinical studies and to work on research projects. Short courses for graduates and "continuation courses" have been established in a number of medical schools, primarily for doctors in practice. Recently societies of specialists have established courses of didactic instruction, forums and round table discussions designed primarily to make available to the practitioner who cannot take a course in an institution of learning a comprehensive review of the basic sciences and new developments in the fields of diagnosis and treatment of disease.

It is readily apparent that the burden on our university medical schools can be lightened only by shifting some of the load to other sources of instruction.

Instruction Courses were initiated by the American Academy of Ophthalmology and Otolaryngology in 1921 at the request of members who petitioned the Academy Council to present lectures on selected subjects by outstanding men in medical circles. These lectures were not to supplant the regular program of scientific papers and projection demonstrations, but they were to supplement the subjects considered at the annual convention.

Immediately the Instruction Courses in Ophthalmology and Otolaryngology and Maxillofacial Surgery became popular with the members and teachers as well. The management of the courses was entrusted to a member of the Council, who later became known as the Secretary on Instruction, and more recently to a Secretary on Instruction for Ophthalmology and a Secretary on Instruction for Otolaryngology and Maxillofacial Surgery — both members of the Board of Secretaries of the Academy.

The courses were developed by natural growth rather than through planned direction. Subjects were selected by request of members or on the suggestions of instructors.

The popularity of subjects and of instructors is judged by the attendance, and changes are made to best meet the interests of the teaching program. The value of these lectures can be judged by the continued interest found in local, county and district societies. Usually two or three members of a local society are designated to report to their meetings throughout the year on the lectures they have attended at the annual convention of the Academy. More than seven hundred hours of instruction were offered by a faculty of 375 during the five days of instruction at the annual convention in 1954, and total hours attendance was more than 16,600.

The success of the Instruction Courses stimulated another project that has been in continuous operation since 1940. The Home Study Courses in Ophthalmology and Otolaryngology were designed to aid fellows and residents below the Board level in a planned re-

view of basic subjects. Running for 10 months, they are conducted through the home office of the Academy and are supervised by a member of the Secretarial Board. A faculty of about fifty is selected from the membership of the Academy to prepare reading references on a fundamental subject such as "physiology." A different subject is selected for each of the 10 months. Subscribers to these courses spend an average of two hours a day for one month on the reading references. They are then sent a list of twenty questions on the subject. The written answers are referred to the faculty for comment and returned to the student. This is not an examination, and the papers are not graded on a percentage basis. No certificate is issued nor Board credit given. While the Boards of Ophthalmology and Otolaryngology advise their candidates to take the Academy Home Study Courses, results are not reported regularly to the Boards. Home Study Courses are particularly helpful to residents in hospitals with small teaching staffs but are used in nearly all medical schools and hospitals in the United States and in many abroad. Several institutions pay the fee of twenty dollars for each student. The total enrollment in Home Study Courses averages about 400 per year. The Academy supplements the Instruction and the Home Study Courses by publication of about twenty teachers' *manuals* written especially for their courses.

The Academy has published two pathological atlases prepared with cooperation of the Armed Forces Institute of Pathology, and has produced two motion pictures in color with sound: (1) Embryology of the Eye, and (2) Embryology of the Ear, supplemented by lantern slides and made especially for teaching. More than 40 copies of the film Embryology of the Eye, produced in 1952, are now owned by medical schools. The Embryology of the Ear will be released early in 1955.

This brief recitation of the activities of the Academy illustrates the opportunities that exist in the societies of medical specialists for self help and the advancement of medical education in both fundamental and clinical subjects to their colleagues and to future specialists.

Many of the features of the Academy program of graduate education and instruction are adaptable to other specialties. In fact, several societies recently organized have been patterned after the Academy plan and have prospered. The Instruction and Home Study Courses are inexpensive and self supporting. They are conducted by physicians for physicians with mutual benefit.

W. L. Benedict, M.D.

INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

ASSIGNMENT — INTERNATIONAL HOSPITALITY

Acting as hosts, welcoming guests in the foreign visitors' lounge at the XVII International Congress of Ophthalmology, was the rare privilege afforded members of our organization. At this congress which met in New York City, September 12 to 17, 1954, we greeted colleagues from many lands. All five continents were represented.

It was indeed a thrilling and stimulating experience to enjoy the atmosphere and complete good will and mutual trust which prevailed among this heterogeneous group. If the world's political leaders could be motivated by this same wish to understand each other, peace and harmony would dispel hatred, suspicion and unrest, and happiness would become the common denominator of all mankind.

The officers of the American Academy of Ophthalmology and Otolaryngology, as is their custom, again invited us to be their guests at a dinner meeting. The many foreign representatives present added much to the festivity of the occasion by their general enthusiasm and were most appreciative of the hospitality shown to them.

Now to say a word about us here at home. If our directors will assume a more active part in helping to stimulate interest in the various local and state meetings, this will be of tangible assistance to all the secretaries in their unremitting efforts to provide fine programs.

So, let us remember that each member of our organization has been asked to assist at the meeting of the Sixth International Congress of Otolaryngology to be held in Washington, D. C., May 5 to 7, 1957. Based on our recent most pleasant experience in participation at an international meeting, we look forward to this event and our part in it with a keen anticipation.

RODERICK MACDONALD, M.D.
President

OPPORTUNITIES UNLIMITED

The world is hungry for scientific knowledge.

Ophthalmologists and otolaryngologists in every land are asking for the know-how, the skills, the tools, the techniques, and the teaching methods which will help advance their local societies.

The International Association of Eye, Ear, Nose and Throat Society Secretaries with a membership of over 249 societies in 42 countries on five continents is beginning to share this accumulated information.

The task before us is a real challenge and a great opportunity.

The 1954 annual meeting in New York City was enthusiastically attended by fellow secretaries from every section of America, and from more than a score of foreign countries. Momentum is gathering. The year 1955 promises to bring together an even greater number of colleagues from distant lands.

Every effort will be made to facilitate the bringing together of secretaries, in order to share scientific knowledge and thereby encourage international friendship. Underdeveloped countries are looking to us for help. Each country can make some contribution to the world's reservoir of knowledge. There are no limits to the benefits and goodwill which can result from this free exchange of ideas and methods.

Your officers will welcome your suggestions and comments in planning for the future.

WILLIAM F. GOFF, M.D.
President-Elect

OTOLARYNGOLOGIC RESIDENTS AND THE SCOPE OF TRAINING

LESTER A. BROWN, M.D.
ATLANTA, GEORGIA

I should like to speak briefly concerning two topics: first, on the subject of otolaryngologic residents, or the lack of them; and second, on the controversial subject of what the members of our teaching staff are learning from the course of training that is being offered to the residents.

By way of clarification I might say that although I give some of the lectures in otology to the medical students of Emory University in Atlanta, my primary assignment with that medical school is that of supervising the E.N.T. residency training program at the Veterans Administration Hospital, which is under the Dean's Committee. My so-called living comes from private practice. Next, I have no idea of presenting anything new. Third, my remarks are in no way a criticism of anyone. Fourth, my observations are confined to conditions present in my own situation. Fifth, there are no full-time men in our E.N.T. department other than the residents. Sixth, as I use the pronoun "we," I am referring to the collective opinion of several members of the teaching staff.

No doubt, the methods which offer the best prospects for procuring residents can be summarized in one statement: interest in otolaryngology can be developed only by association. Perhaps this is not specific to our specialty.

There are three levels at which we might hope to interest men in otolaryngology: the medical school level, the first year internship level, and the general practice level.

It would seem that four years in medical school would be adequate for training in the specialties in addition to the general subjects; however, this is a very short time, which appears long only to three persons, the college student who is preparing to go into medical school, his wife, and the father who will have to pay for these four years. Nevertheless, we may accomplish something in this limited period if we can acquaint the student with our instruments, our methods, and the members of our staff, convince him that the cavities of the ear, nose and throat are as accessible for examination as are the body surfaces, and prevail upon him to include a good ear, nose and throat examination in every general physical examination. The more opportunities we have to present our work to the student, the more chance we have of gaining his interest, then and later. Maybe what he doesn't know will not hurt him, but it will hurt us. The medical school level is the important one.

At the second level, that of the first year internship, there are relatively few conferences in otolaryngology as compared to those in general medicine and surgery, which is partly the fault of the specialist and is partly due to the lack of interest of the intern. This means that the neonatal physician comes to know the internist and general surgeon

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From the Department of Otolaryngology, Emory University School of Medicine.

and their trades better. It is difficult to interest the intern in a type of surgery that affords little more room and visibility than is necessary for the operator; but, there arises a question as to whether the widening of the scope of E.N.T. by such methods as taking on cancer of the head and neck would actually gain interest in the basic principles of otolaryngology. This situation can be helped somewhat by the specialist taking adequate time and interest in going over each consultation thoroughly with the intern, by no means neglecting to become personally acquainted with him, for it is the amount of skill and interest manifested by the specialist which causes the intern to feel the importance, or lack of importance, of our work.

The third level, that of the young man in general practice, is the source of most of our residents, men in their late twenties and early thirties. Usually, these men have given this matter some thought during the first and second levels, through association with an instructor, a relative, or a close friend, so it is important to keep in touch with them after they leave internship. Further, when an otolaryngologist recommends a young doctor for training, that doctor gets the next available resident's place, and under the present conditions, the places are available. Doctors coming into the specialty via this route make good. In the eight years that I have supervised this residency program, no one of these men has failed to complete his course of training, no one has changed his specialty after completing the residency, and no one has gone broke after going into the private practice of otolaryngology or the combined practice of otolaryngology and ophthalmology. Not uncommon is it for the otolaryngologist not long in practice to begin to look about for an associate. When I view the specialty from this angle and then hear someone say that otolaryngology is dying, I cannot help but think that it must be sick in a strangely healthy sort of way.

The second, more controversial, subject refers to what the members of the teaching staff are learning from a hindsight perspective of the course of training which is being offered to the residents. Our outline of study includes the usual topics, headlined by gross anatomy, in which the residents act as instructors to the freshman medical students. In addition, a course in public speaking in encouraged, and, off the record, two residents have taken Dale Carnegie's course.

Clinically, we have broadened out to do surgery of benign and malignant lesions of the head and neck, exclusive of the brain and the thyroid gland. Early this year, we undertook to evaluate the work of the E.N.T. department for the past seven years, particularly as related to cancer. Several things became noticeable: (a) cancer patients may have long hospital sojourns, (b) many of the cancer patients die of their immediate disease, (c) of those who survive, the morbidity is high, even to the extent of incapacitation for work and usefulness in some cases, (d) as the proportion of cancer training goes up, the proportion of pure E.N.T. training goes down, especially when the number of beds is limited to between 12 and 15.

As more attention has been paid to the broadening aspects of the specialty, less attention has been paid to such things as the lowly (although breadwinning) tonsillectomy, the submucous resection, the caloric test, the deaf person. Also, we realized that cancer patients were occupying many of the beds, occasionally as high as 70 per cent of them. At the same time, the waiting list for the more common E.N.T. surgical cases was increasing. Keeping these things in mind, we decided to follow

the pendulum from its lateral to a more medial position. In other words, we would continue with the surgical treatment of malignant lesions of the larynx, the sinuses, the ears; continue with the neck dissections where indicated; but make no effort to take over the patients who had other malignant lesions of the head and neck. Restricting cancer work has given us additional bed space for treatment of the more common diseases of the ear, nose and throat, and the beds continue to be occupied.

With this change, members of the E.N.T. resident staff seem more interested in their work. Perhaps this arises, to some degree, from a coincidental situation which is universally pleasing to E.N.T. sections; that is, the almost total segregation of the service (and being from Georgia, I use this word very cautiously, with no idea of opposition to the Supreme Court). In the next few years, we may find that making these narrowing changes was not wise; but now we feel that it might be good judgment to spend more time with diseases of the ear, nose and throat. There is no argument about the cancer patient needing help, but help is coming by way of the cancer clinics; therefore, as we reflect upon one of Doctor Mosher's many famous expressions, that "one should endeavor to do some one thing better than anyone else can do it," might we profit by not expanding too broadly?

Summarizing: First, since it is our feeling that procuring residents can be done only through association, we are making some attempt at intramedical advertising at all three levels. Second, we are inclining a bit to the purer otolaryngology and, to some extent, drawing in our branched tentacles.

In conclusion, it occurs to me that I have never thought of otolaryngology as being high on the list of specialties for drawing residents; but the history of otolaryngology is long, and I am firmly convinced that there will always be enough peculiar doctors who will want to go into otolaryngology, and there will always be enough people with peculiar complaints to keep them busy.

SHORTAGE OF CANDIDATES FOR OTOLARYNGOLOGIC RESIDENCIES

JAMES W. MCLAURIN, M.D.
NEW ORLEANS, LOUISIANA

All of you know why it is necessary for us to discuss at this meeting how we shall go about finding residents in otolaryngology. The blunt truth is that a large proportion of the available residencies have gone begging for the past several years, and they are still going begging.

A discussion of this unhappy situation can be profitable only if we begin with the confession of past errors. I do not know, as the General Confession puts it, how many things we have done that we ought not to have done, but I know very well, as I am sure you do, that we have left undone a great many things that we ought to have done.

Foremost among our errors of omission is our complacent acceptance of the fact that in most institutions in the United States and Canada this specialty still does not have its proper place in the sun. In some institutions, in fact, it has, for all practical purposes, no place at all.

You are familiar with the reports in which these details have been pointed out. Students are not likely to think it necessary to acquire specialized training in a field of which medical faculties think so little that they allot such limited

From the Department of Otolaryngology, Tulane University of Louisiana School of Medicine.

amounts of time to it in the undergraduate curriculum. Small wonder that later, if they do wander into this field, many of them become what a distinguished New Orleans gynecologist was, I believe, the first to term "self-appointed" specialists.

It is not an easy matter to alter this situation. I speak as the veteran of one such battle. Up until 1949 the entire course in otolaryngology at the Tulane University School of Medicine was taught in the fourth year. Instruction was limited to six hours of didactic lectures and twelve hours' work in outpatient clinics. It was obviously impossible to teach very much, let alone arouse an interest in otolaryngology, in such a limited amount of time. When I became responsible for instruction in the department, I set out to alter the current arrangements. This was not easy. The authorities were sympathetic. For that matter, I had considerable sympathy with them. I was not the only department head who was demanding more time. Many months were to pass before the battle was won, or, to be more accurate, partially won.

Medical students at Tulane are now introduced to otolaryngology in their second year. Their chief instruction comes in the third and fourth years. Elective courses are offered in the fourth year. We still have very far to go before we reach the level recommended by the committee which, to save breath and space, I shall merely describe as the Committee on Undergraduate Teaching of the Academy, but at least we are now graduating students who realize that otolaryngology is a specialty and not something practitioners of medicine are born knowing about.

You will remember that this committee recommended two hours of orientation and four of physical diagnosis in the second year, and 40 hours of didactic and clinical instruction, preferably in the third year but arranged according to the facilities available and the discretion of the head of the department. Electives occupied the assigned hours in the fourth year. I hope that we shall eventually come to this at Tulane.

The committee also recommended that otolaryngologists teach the anatomy of the head and neck in the first year. Here I do not go along with the recommendation, though I think it would be highly profitable for otolaryngologists to be allowed one or two sessions with each group as it completes this particular phase of the course. The lectures should be entirely practical and should relate the regional anatomy to clinical problems. The student will have a much better understanding of both anatomy and otolaryngology if he begins to learn, early in his course, that anatomy is something more than an academic requirement and that diseases of the ear, nose and throat are sufficiently numerous, varied and serious to require their respectful attention. Many medical students come from small towns, where the general practitioners, as a matter of necessity, practice otolaryngology, and they cannot learn too soon that this specialty involves something more than upper respiratory infections and tonsil operations. The sooner medical students begin to make contact with clinical medicine in all fields, the better for them and for their future patients.

Similarly, I should like to see the otolaryngologist participating in the course in physiology. Discussions with students, and even with residents, sometimes suggest that it is not entirely clear to them that the ear, nose and throat have any physiologic functions. I should also like to see the otolaryngologist participating in the teaching of pathology. He has a good deal to contribute to

clinicopathologic conferences. Specimens of considerable interest are often available from the ear, nose and throat, and patients do die of disease originating in these areas.

Dr. Russell Decker writes me that at the University of Southern California otolaryngologic teaching is concentrated in the fourth year, during which each student spends a three-week period on the service, attending formal lectures, clinics, ward rounds, staff conferences and seminars, elective and emergency operations, and anything else that concerns the department. In addition, each student spends one day of each of his three weeks with a member of the staff, attending his operations, seeing patients in his office with him, and receiving the invaluable experience that was lost when the preceptor system was discarded. I can easily understand why, as Dr. Decker wrote me, the students "seem to get a big kick out of these days." I am also impressed by the excellent effect on residents and interns of having to make ward rounds with students on the service twice daily.

The demands otolaryngologists are making on the student's time are not at all unreasonable. We are merely asking for enough space in the curriculum to instruct him in normal anatomy and physiology of the nose, throat and ear; to teach him methods of physical examination and the use of the simpler instruments of diagnosis; to provide enough didactic and clinical instruction to make him realize that an otolaryngologic examination should be part of all physical examinations; to teach him to recognize abnormalities when he encounters them; and to show him, by precept and example, how to deal with patients with diseases of the ear, nose and throat.

The faculty of any department of otolaryngology, continuing to make up for past errors of omission, must make a determined effort to interest themselves in what the student is interested in, though this error of omission, I hasten to add, is not confined to our branch of the teaching profession. I think we have lost a considerable amount of our potential influence over all students by failing to do this. We should attend their meetings when we are invited to them. We should also, when we are invited, attend their social functions, or at least see that the department is represented at them.

Elective courses furnish excellent opportunities for making personal contacts with students. These courses are seldom overrun with candidates. The students who elect them are showing at least a glimmering of interest in becoming otolaryngologists. We should carefully nurture that glimmer. Elective classes should be made a great deal more than perfunctory performances. I was much interested in Kirchner's experience of three theses in otolaryngology at Yale one year and seven the next. I have no doubt that he is correct in his assumption that the increase came because he had the time the second year to devote to student interests and problems.

Last year, for the first time, a cash prize was offered at Tulane to the student who submitted the best thesis in otolaryngology. It attracted attention, and as time passes I am sure it will attract more. We made the prize one hundred dollars, and I shall not surprise you when I add that I am sure that the cash rather than the abstract glory attracted some students to enter the contest. The end result was what we wanted, however: more students were interested in otolaryngology.

Otolaryngologic teaching is, of course, not going to be what it should be unless the department is autonomous. The bad

name sometimes attached to the specialty can be attributed, in part, to the fact that it is often an appendage of the department of surgery. The old concept that it is limited to the performance of tonsillectomies still sticks. As a matter of fact, otolaryngology is both a medical and a surgical specialty. It functions best when it is attached to neither medicine nor surgery. It is most efficient when it works in cooperation with the department of surgery, including plastic and chest surgery, and with the department of medicine, including allergy. Cooperation between the department of otolaryngology and the department of roentgenology is, of course, essential. A practical means of cooperation is the assignment of men from the department to the other departments for special integrated teaching. I will not deny that the opening wedge takes quite a bit of doing, but the present tendency to cross departmental lines in teaching is all in favor of such a plan.

One other point might be made about an autonomous department. It can provide the best quality of teaching, because it can get and keep good men. The best qualified men are not going to be happy in a department which functions as part of either medicine or surgery, and they will solve that particular problem by doing half-hearted teaching or by leaving the department altogether.

Having gotten our man out of the medical school aware that there is a specialty of otolaryngology, what comes next? If it can be arranged—and it usually can be if the otolaryngologists are positive enough about it—the rotating internship should include a period in otolaryngology. Patients with diseases of the ear, nose and throat receive better care under this arrangement, which also, as Friedberg well puts it, offers opportunities for the "recruitment of the undecided." As he has pointed out, half or more of all interns have no idea what they are going to do or become when they begin the intern year.

These opportunities are frequently mishandled or overlooked entirely. Friedberg, you may remember, in an analysis of rotating internships in teaching hospitals approved for training, found that intern exposure to otolaryngology occurred in not quite 75 per cent of the total number which replied to his questionnaire, and that exposure could be regarded as adequate in only about half of the 75 per cent.

This is another area in which we have erred greatly in the past. The perfunctory accomplishment of internships in otolaryngology, with supervision by residents, on sloppily conducted wards on which a senior visiting man is seldom seen, does us no good. Whether a formal program of intern instruction is necessary I am not yet prepared to say, but I know that a great deal more instruction should be given on rotating internships than is now given. Priest, last year, mentioned a manual of procedures on ear, nose and throat work which had been prepared for intern use at the Minneapolis General Hospital. I should like to know how it has worked out. It may prove to be one more way of interesting the intern in this field.

Then, having, let us assume, caught a resident, we must give him the same amount of attention I have been pleading for in his undergraduate and intern years. I felt that we had made a great step forward at Tulane when we began to combine the basic science requirements with clinical training instead of concentrating them in a single nonclinical year. Our objective must always be to keep the resident and the patients together. He must be given the responsibility for a large amount of work, both surgical and nonsurgical, as

his training proceeds. If the attractive cases are all pre-empted by the senior men, the resident will, justifiably, have a grievance. He will express it by doing poor work and by downgrading the service in his contacts with interns and students.

What I have said does not, of course, mean that either residents or interns should have a free hand. My own rule at Tulane is that there be no unsupervised work in the department. Each clinic and each operating section is attended by a member of the staff. Ward rounds are frequently made. The staff knows what the residents are doing. My own feeling is that when this policy is clearly enunciated and strictly followed, the feeling between staff and residents is a good deal better and the residents have a good deal more respect for the staff and for their own training.

One other error should be stopped at the source, the talk about otolaryngology as a dying specialty. I think it regrettable that the Academy or some other official organization did not formally register the disapproval of the membership when the editor of the *Journal of the American Medical Association* took occasion recently to inform us that ours was a "shrinking surgical specialty, subject to encroachment, modification and erosion from various sources," and hemmed in by various competitors from other specialties. The author of these remarks must have been taught otolaryngology rather badly if he has the impression that even in the days of too many tonsillectomies this was ever a purely surgical specialty.

For my own part, I do not feel in the least fenced in. The field of otolaryngology has greatly expanded since my own student days. Endoscopy has become a subspecialty. Allergy has assumed increasing importance. The whole field of audiology, including the fenestration operation on one hand and aural rehabilitation on the other, has opened up. I do not know of any more rewarding work than to bring people, adults and children alike, back into contact with the world by correcting their handicap or teaching them to compensate for it. Infections of the ear, nose and throat have not been eliminated by chemotherapy and antibiotic therapy. Those of us who treat children with chronic middle ear disease or impaired hearing resulting from the unwise use of those agents could wish that otitis media were being treated by more surgery, not less. Myringotomy is still a useful operation.

The most ironic thing about the shortage of residents in otolaryngology is the wide demand for otolaryngologists all over the country. Physicians in private practice are looking for assistants because their offices are crowded. I don't know any competent otolaryngologist who is not making a good living and who does not have more work than he can possibly handle. As for full-time professorships and other teaching positions, they are going begging. It would be a good plan, which I think is seldom followed, to sit down with possible candidates for residencies and discuss with them what the prospects are for the future, once their training is completed.

Lederer, some years ago, pointed out that eye, ear, nose and throat medicine had been regarded as quackery and left to quacks until about 1800. Even in the middle of the last century Abernethy held that point of view. When he was asked to serve as Honorary President of the London Eye and Ear Infirmary, he was perfectly explicit in his refusal. He could see no good that could arise for the public, he said. "I candidly tell you I consider it quackery," he went on. "I will never lend my name to sanction it."

Today nobody considers the specialty quackery. On the other hand, I do think that we often fall into the error of apologizing for it, and doing what, in my part of the South, we call "making a poor mouth" about it. Otolaryngology can survive on its own merits. I see no reason for changing its name. I see no reason for compromising with other specialties. I see, however, a great need for a change in our performance as it relates to undergraduate teaching, internships and residencies.

I don't think, in this informal presentation, that I have introduced very much that is new. I know, in fact, that I have borrowed a good deal from what has previously been said on this subject. I do feel very strongly, however, that overcoming shortages in residents in otolaryngology rests with us, and I believe that some of the methods that I have suggested are worth trying. We have talked about some of them a great deal, but very few of us have made any real attempt to try them, beginning with the undergraduate medical student, who is our ultimate source of supply.

THE THREE-YEAR RESIDENCY PROGRAM

EDMUND P. FOWLER, JR., M.D.
NEW YORK, NEW YORK

Your chairman has asked this panel to make a few remarks concerning its feelings about the three-year residency program, which was started in otolaryngology a few years ago. Evaluation at this time makes it clear that the three-year residency is here to stay and is essential if otolaryngology is going to compete with other specialties. Our residents must be as well trained in general medical subjects as the men on the other services; they must be *better* trained in endoscopy, in the care of acute respiratory obstruction and, above all, in examination of the ear, nose and throat of adults and children. Ear, nose and throat men cannot insist upon the best care of patients, especially those seen in consultation with men in general medicine and surgery, and those in plastic surgery, chest surgery and the like, unless they too have at least three years of residency training in addition to a firm grounding in general surgery and general medicine. Preferably, they should have at least two years of training before specializing in ear, nose and throat work. If the medical internship contains some general surgery, this should be quite as valuable as two years of surgical training. The latter usually is predominantly abdominal surgery, with a strong emphasis on the extremities, the heart, or what have you? General medicine can be of much more value to a good ear, nose and throat man.

After the three-year residency, if a man expects to go into a special branch of ear, nose and throat work, such as head and neck surgery, fenestration surgery, or audiology, he should have at least one year more in this special training. This is a long time to be a student but, as you see, I favor more than three years of training rather than less.

Perhaps the E.N.T. residency could be a little more flexible. If a man has had two years in general surgery or general medicine, rosters and ladders might be arranged so that, other things being equal, he could finish E.N.T. in two years, but I doubt if this is feasible, except for, perhaps, the surgically trained man whose interest is primarily in the surgical branches of our specialty and when extra time is taken in plastic or head and neck surgery. The three-year residency assumes that a man will have graduate graded work and enough

From the Department of Otolaryngology, Columbia University College of Physicians and Surgeons.

work. In our hospital we believe that didactic teaching and the basic science course should go hand in hand with practical work on patients. When a man has come out of one of the postgraduate courses, he has had a little more book learning than his colleagues, but in the long run he, as a rule, needs three years of practical training anyhow. In my opinion, adding the graduate course of six or nine months simply lengthens the training period and increases the expense. This hardly seems justified when the extra training can be squeezed into the afternoons and evenings during the residency.

As I see it, the most serious hazard to the three-year residency and good teaching in the future is the growth of the insurance schemes, which insist that every patient must have his own private doctor. This, of course, is perpetrated by the county medical societies and can be pushed too far where teaching hospitals are concerned. At present, with 100,000,000 people in the country insured, some formula must be found which permits the resident to become the patient's "private physician." If the present trend continues, the way certain insurance companies and county medical societies would have it, changes will have to be made in the wording of our present three-year residency setups. This is the way I see the situation in New York City, and I believe the situation is the same along the entire eastern seaboard.

RESIDENCY TRAINING FROM THE VIEWPOINT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

DEAN M. LIERLE, M.D.
IOWA CITY, IOWA

Your chairman, Doctor Hoople, has suggested that I make comments concerning the requirements of the American Board of Otolaryngology and to further relate to you some of my impressions of the fitness of candidates who have appeared for the examination of the American Board of Otolaryngology. I would hope that my remarks are to be construed as constructive. The majority of candidates come from institutions represented by members of the American Academy of Ophthalmology and Otolaryngology, and these criticisms, favorable or unfavorable, may be helpful in the future in evaluating your own residencies.

Shortly after World War II, the Board realized that consideration had to be given to revamping and re-emphasizing the requirements of the Board, both as to the length of residency training and particularly to defining the scope of the field. Reports emanating from various agencies revealed that the young men being certified were inadequately trained. This seemed to be neither the fault of the candidate nor of the institution at the time of certification. There were a number of factors responsible for this:

1. Antibiotics and chemotherapy have played an important role in all fields of medicine. Alterations in the medical and surgical treatment of inflammatory diseases of the sinuses, pharynx, larynx, neck and ear, especially where their complications were concerned, have been accomplished to the betterment of surgical practice. There actually has been little change in the scope of the specialty since the Board was first organized. Because of the abundance of work formerly available, most otolaryngologists were not, or could not be, interested in the broader concepts of the field such as our leaders envisioned for us.

From the Department of Otolaryngology and Maxillofacial Surgery, State University of Iowa College of Medicine.

2. Ophthalmology was separated from otolaryngology as a practical expedient, thus somewhat restricting the field. However, it is to be noted that both fields still occupy the attention of many specialists throughout the country.

3. General practitioners, in many of the rural areas in particular, assumed responsibility for some of the practice always considered to be in the domain of otolaryngology. Today, in many geographical areas, they remove tonsils, treat acute upper respiratory infections, do myringotomies, and unfortunately carry along patients with symptoms and signs of otolaryngologic problems.

4. The chest surgeon began to do his own endoscopy.

5. Military service has created uncertainties of career planning.

6. World economics has curtailed postgraduate training.

Obviously something had to be done (and this still remains a problem) to correct the complex situation. We have chest, orthopedic, urologic, neurologic, abdominal and gynecologic surgeons, so why not otolaryngologic surgeons, who by definition and training do head and neck surgery?

The Board felt that the period of residency training should be increased from two to three years for the following reasons:

1. A three-year period is essential to cover the entire field adequately.

2. For many years we had not met the minimum requirements of a three-year residency recommended by the Advisory Board. We were the only surgical specialty, excepting ophthalmology, which had not required three-year residency. Ophthalmology has since enforced this requirement.

3. The tempo of training has been slowed. Years ago most residents were single. Most of us remember the custom of working on a service until ten or eleven o'clock every night except Sunday. Now practically all residents are married and have obligations of family life, economic as well as social.

What are the present requirements of the American Board of Otolaryngology as far as time required for training is concerned?

1. A candidate must have had a general internship in an approved hospital of at least a year.

2. A candidate must have had training in the basic sciences.

3. a. He must have had a three-year residency or fellowship in otolaryngology approved by the Residency Review Committee of the Board, the A.M.A. and the College of Surgeons.

   b. A four-year residency in combined otolaryngology and ophthalmology, provided one half of the training is in otolaryngology.

   c. The student may take a two-year approved residency and take a year in medicine, surgery, or an extra year of rotational internship in lieu of the third year in otolaryngology.

   d. The Board very wisely left the door open to those individuals who are not able to take formal training. Those who have had an approved internship and training in the basic sciences, and have limited their practice to otolaryngology for seven years, are eligible to take the examination.

e. The Board further states that in exceptional circumstances, candidates that can not meet the above requirements may be accepted for examination upon recommendation of the Credentials Committee substantiated by action of the Board.

I believe that I can bring out more clearly the necessity for three years of residency training by outlining briefly the requirements now as to the scope of the average resident's training. He must have fundamental training in general medicine, surgery and otolaryngology. He must be encouraged to read the current literature and present papers. He must demonstrate proficiency in the applied basic sciences fundamental to the intelligent practice of otolaryngology. These include anatomy of the ear, pharynx, larynx and tracheobronchial tree; nervous system; gross pathology; histopathology; bacteriology; physiology; the fundamentals of otolaryngology; the principles of surgery, and the essentials of acoustics. He must have training in all phases of otolaryngology, peroral endoscopy, maxillofacial surgery, surgery of the neck and audiology.

I wish to emphasize the fact that this three-year period of training, in my opinion, is basic and is designed for the general practice of otolaryngology. Those who have the interest and aptitude may go into special branches of our field which require additional training. I have often heard the remark that otolaryngologists cannot possibly encompass in their practice the entire field. This is quite true, but each otolaryngologist does need the fundamental knowledge in all of it for general practice in otolaryngology.

It is true that we have too few otolaryngologists, but there is a dearth of candidates in other specialties, too. Reasons for the dearth in our specialty, and in my opinion they affect all other areas of medicine and surgery, are the requirement of the armed services, the trend toward general practice, and last, but probably the most important, the economic situation of the young physician after he has completed his internship. The period of required training should not affect us any more than any other specialty.

The economic situation eventually will be corrected when the various institutions discard their antiquated system of inadequate remuneration of residents. How can anyone justify not granting a living wage to any apprentice? A resident who has spent seven to eight years in college is entitled to earn more adequate support in his second year of postgraduate work than he does at present at many institutions. Teachers can do much more to attract men in our field by making their undergraduate and graduate courses more attractive from every point of view.

I would now like to make a few comments concerning the Board examination itself.

Although it is relatively early to draw definite conclusions regarding the three-year residency group, the percentages of conditions and failures are much greater among the indifferently trained residents, less in the two-year group, and least of all in those who have had three years. Generally speaking, however, there is a marked improvement in the fitness of all candidates. This is evidenced by the fact that the percentage of failures and conditions during the past five years in formally trained individuals has dropped very materially. The percentage of failures in the indifferently trained group is much higher than in those who have had formal residency training. Much progress has been made in the last three years in standardization of the scope of training. Each

candidate for the Board examination is asked to give the number and type of surgical procedures he has performed and also the number and type in which he has assisted. The last examination revealed that among 58 candidates from 56 institutions, 49 had some training in maxillofacial surgery; 46 had training in tumor surgery, including neck surgery; and all had endoscopic training. Most of their maxillofacial surgery experience consisted of rhinoplasties.

A great deal more is desired in the way of training in this field. In my opinion otolaryngologic services should have trained plastic surgeons attached to their services, or the aid of the plastic surgeons should be enlisted to permit selected otolaryngological residents to spend adequate time on their services. This would accomplish a great deal in revealing the necessity for adequate training for those individuals who wish to include this work in their practice.

It is also my opinion that more adequate training in neck surgery is needed in many institutions. This may be accomplished by enlisting the aid of the general surgeon or by training teachers in otolaryngology in this particular phase of our work. Many of us have blamed the general surgeon for his unwillingness to share neck surgery. One can find little fault with him if our representatives are not qualified to do this type of work. Certainly more is required than just to say this work is within our scope and domain, and therefore we should do it.

One of the most outstanding and glaring deficiencies observed in the training of residents is in otology. Many candidates even lack training in the basic knowledge in this area. Audiologists in some institutions have done such a thorough job that many residents are relieved of performing the duties of routine hearing tests. As a consequence some individuals are unable to do audiometric and speech tests or to interpret audiograms, and they know little about fork and vestibular tests. There are some that are unable to visualize perforations and lack the ability to interpret pathologic lesions in the middle ear. Some are even unable to determine correctly the type of mastoid operation that has been done, or to recognize the indications for such an operation.

Certain candidates are particularly weak in the interpretation of x-rays of the sinuses, mastoids, skull, cervical vertebrae, and the chest. By all means, they should have more information than they do in basic knowledge of x-ray and radium therapy.

Many of the candidates are deficient in their knowledge of the basic sciences. Their information is too general, and they lack specific knowledge. The Board has attempted to correct this situation by having a separate qualifying examination in the basic sciences by a special group of responsible examiners.

Some years ago candidates had little knowledge of histopathology, but since the Board placed special emphasis upon this subject, most of the candidates have a good working knowledge of histopathology. Moreover, it must be recognized that the Board's action stimulated a general interest in the field.

Another one of the bright spots in today's training is endoscopy. Here again most of the candidates show evidence of excellent training in this field.

A number of the candidates are not thorough enough in history taking and in carrying out the physical examination. Some are entirely too rough with their patients. Emphasis in training programs should be placed on thoroughness, gentleness, and cleanliness.

In conclusion, there has been a marked improvement in the last five years as far as the candidate's knowledge of otolaryngology is concerned. This is evidenced by a smaller percentage of conditions and failures. More institutions are teaching the entire scope or field of otolaryngology. However, there are still some institutions which are having difficulty, particularly in surgery of the neck and maxillofacial surgery. I am sure that these deficiencies will be corrected in the near future.

The real answer to much of our problem is to develop well trained young teachers. These teachers should have a thorough background in general surgery and medicine. It is also my opinion that they should be encouraged to develop and teach a particular field in otolaryngology, such as otology, tumor surgery, or maxillofacial surgery. It should be made possible for some of these individuals to become interested in research. These young men would do much to help in the training of our residents and in teaching medical students the practical aspects of our specialty. We must all strive to make the teaching of otolaryngology so attractive that we will attract many more young men to enter our specialty. The aims in regard to procuring and interesting more trained young otolaryngologists as teachers can be accomplished. I know of two young men at this time who have several years of training in general surgery and medicine, three years of residency in otolaryngology and several years of teaching experience. These men are excellent teachers and clinicians. They are interested and have done commendable research. Otolaryngology has indeed, in my opinion, a very bright future when young men of such stature become increasingly available.

MEETINGS OF FOREIGN SOCIETIES

THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Date: Annual Congress, April 28 to 30, 1955
Place: London, England
Secretary: Dr. Derek Ainslie
87 Harley Street
London W1, England

SOCIETE FRANCAISE D'OPHTALMOLOGIE

Date: May 8 to 12, 1955, 62nd Congress
Place: Maison de la Chimie, Paris
Secretary: Docteur Marcel Kalt
81 Rue St. Lazare
Paris 9e, France

ENTRETIENS ANNUELS D'OPHTALMOLOGIE

Date: May 12 to 14, 1955
Place: Maison de la Chimie, Paris
Secretary: Professeur G. E. Jayle
91 Rue St. Jacques
Marseille, France

News - - - Notes

PAN AMERICAN CONGRESS OF OPHTHALMOLOGY

The Fifth Pan American Congress of Ophthalmology will be held in Santiago, Chile, from January 9 to 14, 1956.

Twelve symposia dealing with the following subjects have been organized: Collagen Diseases, Glaucoma, Infantile Glaucoma, Secondary Glaucoma, Strabismus, Detachment of the Retina, Psychosomatic Ophthalmology, Tropical Diseases, Physiopathology and Surgery of the Crystalline Lens, Plastic Surgery, Visual Fields and Neuro-Ophthalmology, and Intraocular Tumors. Participants in the different symposia are equally divided in numbers between ophthalmologists from the United States and Canada and from Latin America.

Special provision will be made to enable English-speaking delegates to the Congress to understand papers presented by their Latin-American confreres and vice versa.

For further information please contact Dr. Rene Contardo, 930 Huerfanos, Santiago, Chile.

THE AMERICAN GOITER ASSOCIATION

The American Goiter Association will hold its 1955 meeting in the Skirvin Hotel, Oklahoma City, Oklahoma, April 28, 29 and 30. The program for the three day meeting will consist of papers and discussions dealing with the physiology and diseases of the thyroid gland.

AMERICAN ORTHOPTIC COUNCIL

For the benefit of those ophthalmologists interested in obtaining the services of a qualified orthoptic technician, the American Orthoptic Council has notified us that Miss Laura B. Drye, director of the Confidential Placement Service of the American Association of Orthoptic Technicians, will be happy to be of assistance. Her address is Eye and Ear Hospital, 230 Lothrop Street, Pittsburgh 13, Pa.

IOWA

The Basic Course in Orthoptics for Technicians, sponsored by the American Orthoptic Council, will be held in the Department of Ophthalmology, University Hospitals, Iowa City, Iowa, from June 20 through August 13, 1955. As usual, there will be didactic lectures and practical demonstrations given by an outstanding faculty. Further information, as well as applications, can be obtained by writing to Hermann M. Burian, M.D., Department of Ophthalmology, University Hospitals, Iowa City, Iowa.

NEW YORK

The National Council to Combat Blindness, Inc., announces it is at this time welcoming applications for its 1955-56 research awards for grants-in-aid and full-time research fellowships in the field of ophthalmology and its related sciences.

The Council has added to its research program the financing of summer fellowships initiated by its Scientific Advisory Committee as a stimulus to the development of ophthalmic investigators. All applicants for fellowships, full-time or summer, are required to make their own arrangements for suitable research facilities with accredited institutions.

Applications for 1955-56 grants-in-aid and fellowships will be considered at the sixth annual meeting of the organization's Scientific Advisory Committee, to be held in May 1955. The closing date for the receipt of completed applications is April 15. Appropriate forms may be obtained by addressing the Secretary, National Council to Combat Blindness, Inc., 30 Central Park South, New York 19, New York.

NORTH CAROLINA AND SOUTH CAROLINA

The North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will hold a joint meeting at Columbia, South Carolina, on September 12, 13 and 14, 1955. Headquarters will be the Columbia Hotel.

The following ophthalmologists will be on the program: Dr. E. W. D. Norton and Dr. Frank Carroll of New York, and Dr. William B. Clark of New Orleans.

An announcement of the three guest otolaryngologists who will speak will be made later.

PENNSYLVANIA

The College of Physicians of Philadelphia, Section on Ophthalmology, met on Thursday, January 20, 1955. The Scientific Program was presented by Dr. Jose Fiol-Bigas, whose topic was "Case Report: Hydrops Corneae with Keratoconus"; Dr. Samuel M. Diskan, who spoke on "Visual Screening in Schools"; Dr. Thomas B. Hedges and Dr. Francis Heed Adler, who presented a movie titled "Ocular Motor Manifestations of Central Nervous System Disease"; and Dr. William C. Frayer and Dr. Harold G. Scheie, whose topic was "Coats' Disease—A Clinical and Pathologic Study."

The thirteenth annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held May 18 to 21, 1955, at the Hotel Traymore, Atlantic City, New Jersey. This meeting is being held jointly with the New Jersey Society of Ophthalmology and Otolaryngology and the West Virginia Academy of Ophthalmology and Otolaryngology.

Dr. James H. Delaney, President of the Pennsylvania Academy, will preside. The guest of honor for the meeting will be the President-elect of the American Medical Association, Dr. Elmer Hess.

The meeting will consist of study clubs and general sessions. The faculty for the study clubs will be members of the societies. Papers at the general sessions will be given by members of the societies and invited guests.

CANADA

The Seventeenth Annual Meeting of the Canadian Ophthalmological Society was held at the Physical Sciences Centre, McGill University, Montreal, in September 1954. Doctor Mark Robert Marshall was in the chair.

This being the year of the International Congress of Ophthalmology, the meeting of the Canadian Ophthalmological Society was transferred from the usual meeting time in June to just before the extension of the International Congress held in Montreal on September 10 and 11. On account of this there was a large attendance of visitors. This was the source of great pleasure and satisfaction to the members of the Society.

The Scientific Program was abbreviated and consisted of nine papers. Prominent among these were those of the two guests of honor. Doctor John H. Dunnington of the Institute of Ophthalmology, Presbyterian Hospital, New York, spoke on "Defective Ocular Wound Healing—An Experimental and Clinical Study" and Professor G. B. Sourdille of Nantes, France, spoke on "Lamellar Keratoplasty." The papers of both of these distinguished ophthalmologists will appear in the Transactions of the Canadian Ophthalmological Society.

Doctor Dunnington and Professor Sourdille were elected Honorary Members of the Society.

Doctor Percy B. Macfarlane of Hamilton, Ontario, was elected President for the coming year, while Doctor Henri Pichette of Quebec City was elected Vice-President. The Secretary, Doctor R.G.C. Kelly of Toronto; the Treasurer, Doctor J.V.V. Nicholls of Montreal, and the Editor-in-Chief of the Transactions, Doctor Clement McCulloch of Toronto were re-elected.

EGYPT

The Annual Meeting of the Ophthalmological Society of Egypt will take place at the Memorial Ophthalmic Laboratory, Giza, Egypt, on Friday and Saturday, March 4 and 5, 1955. Medical practitioners, oculists or otherwise, are cordially invited. Dr. Sabri Kamel, of Cairo, is Secretary of the Society.

ENGLAND

The Council of the Ophthalmological Society of the United Kingdom has instituted a prize of 100 pounds, awarded triennially, for the best essay submitted upon a subject selected by the Council, to be known as The Treacher Collins Prize Essay. The prize shall be open to qualified medical practitioners of any nationality. The essay shall be written in the English language.

The subject for the next award of the prize is "Diseases of the Lacrimal Apparatus and Their Treatment."

The closing date for sending in essays for this award is December 31, 1956. Essays should be submitted to the Honorary Secretary, Ophthalmological Society of the United Kingdom, 45 Lincoln's Inn Fields, London, W.C. 2, from whom also any further particulars can be obtained. No name should be on any essay, but a distinguishing pseudonym or quotation, which should be upon a sealed envelope containing the candidate's name and address, should accompany the essay.

IRELAND

The Irish Ophthalmological Society will hold its Annual Meeting in Dublin, May 12, 13 and 14, 1955.

The Montgomery Lecture will be delivered by Dr. D. Arruga of Barcelona, Spain, at five o'clock on Thursday, May 12, in the Royal College of Surgeons, Dublin, Ireland.

PHILIPPINE ISLANDS

At a meeting of the Philippine Ophthalmological and Otolaryngological Society held in Manila on November 26, 1954, Dr. Brittain Ford Payne of the New York Eye and Ear Infirmary was unanimously elected to Honorary Fellowship in the Society. This honor was granted to Dr. Payne in recognition for his invaluable help, friendship and interest in the Society and its members.

POSITIONS AVAILABLE

Otolaryngologist: Diplomate (or eligible) for association with two Board ophthalmologists; Wisconsin City of 28,000. Write: Dr. J. F. Pember, 508 W. Milwaukee St., Janesville, Wis.

Available: Two year residency — ophthalmology, beginning July 1, 1855. Apply: Office of the Executive Director, The Jewish Hospital of Brooklyn, 555 Prospect Place, Brooklyn 38, N. Y.

Diplomate of the American Board of Otolaryngology expects to leave army in July or August 1955. Is interested in establishing a practice in otolaryngology in the Upper Midwest. Fellow of the American Academy of Ophthalmology and Otolaryngology and of the American College of Surgeons. Please write to Major Frederick L. Harcourt (MC), A.P.O. 503, San Francisco, Calif.

Assistant residencies in Ophthalmology available now for July 1, 1955. Expansion in this medical school service with active teaching and research program leads to immediate availability for senior assistant residency leading to senior residency in January, 1956, or availability of junior assistant residency July 1, 1955. Apply: Winston Roberts, M.D., Bowman Gray School of Medicine, North Carolina Baptist Hospital, Winston-Salem, North Carolina.

Fellowship in Allergy and Otolaryngology for a period of three months with a stipend of $200.00 per month. For further information, write to French K. Hansel, M.D., 1229 Missouri Theatre Building, 634 N. Grand Boulevard, St. Louis 3, Mo.

ASSOCIATE IN OTOLARYNGOLOGY. Because of an ambiguity in the wording of a notice appearing in the Nov.-Dec. issue of the Transactions, it appeared that Dr. Milton Jennes was seeking a connection with an established otolaryngologist. This is not the case. Dr. Milton L. Jennes, 14 Central Ave., Waterbury 2, Connecticut, is desirous of securing a well-trained otolaryngologist as an associate in his office in Waterbury.

Assistant Residency in each service—ophthalmology and otolaryngology, beginning July 1, 1955. AMA, ACS approved. 45 bed EENT Hospital. Salary and maintenance. Apply: Superintendent, McPherson Hospital, Durham, N. C.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

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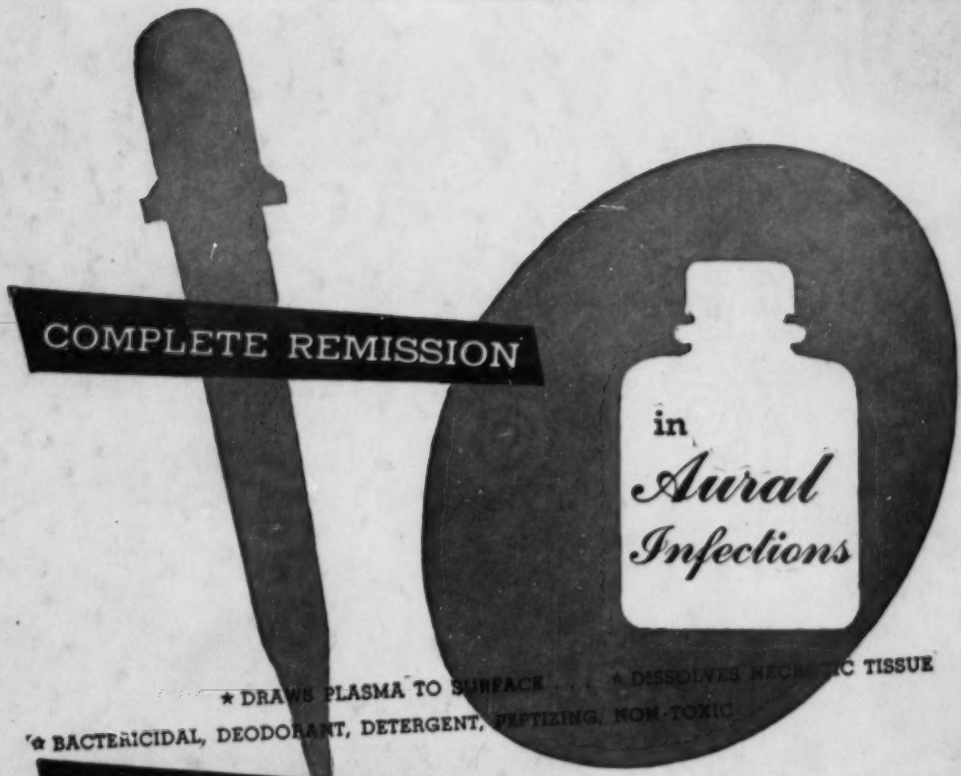
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